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SUTURES IN ANCIENT SURGERY

Alexander Read

(1586-1641)

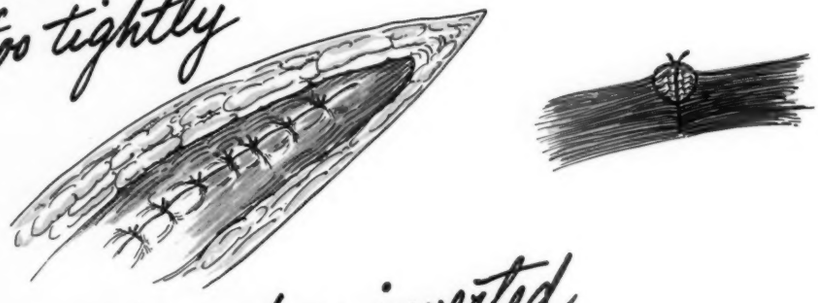
SUTUREGRAM

NUMBER EIGHT IN A SERIES

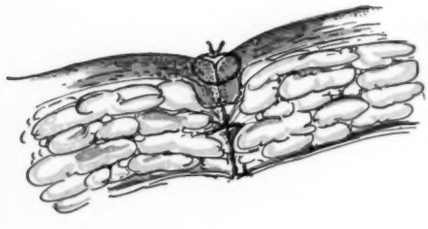
Improper Use of Sutures – Ligatures

1. Large tufts of fat ligated

2. Muscle compressed by sutures tied too tightly



3. Skin edges inverted

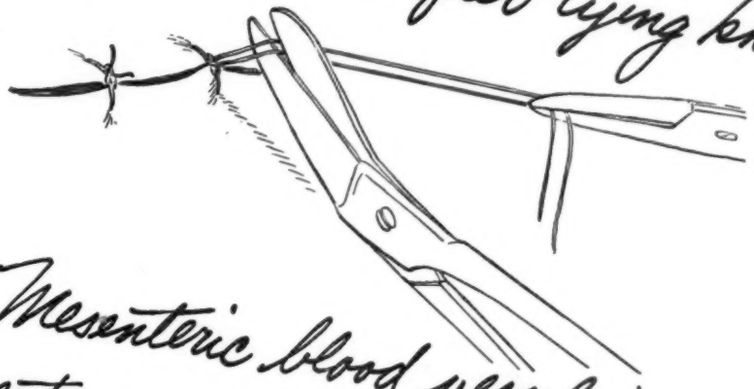


– edges do not heal they separate when sutures are removed

SUTUREGRAM

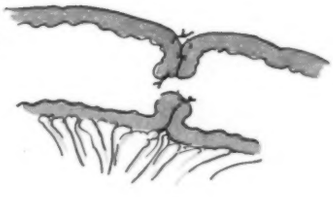
NUMBER EIGHT (CONTINUED)

4. Long end cut after tying knots



5. Mesenteric blood vessel in upper mesentery pierced by needle forming hematoma

6. Too much tissue inverted in an anastomosis
Edema excessive = poor function



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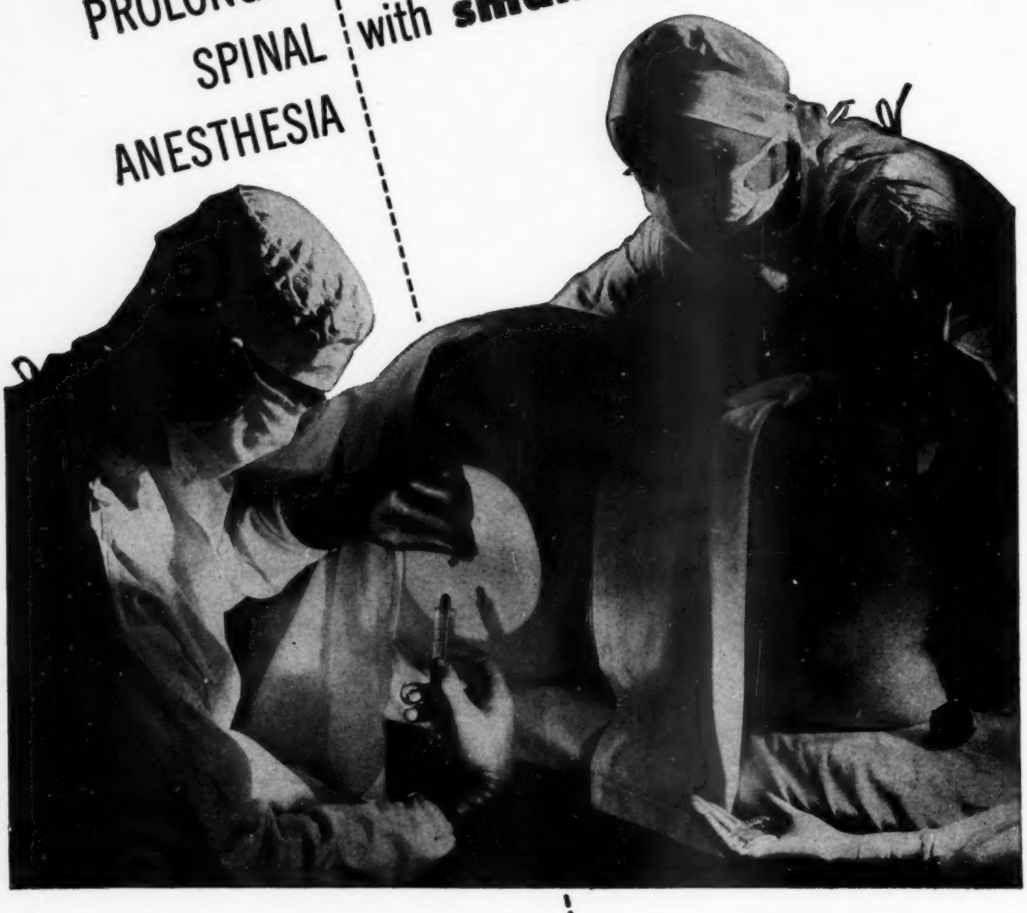

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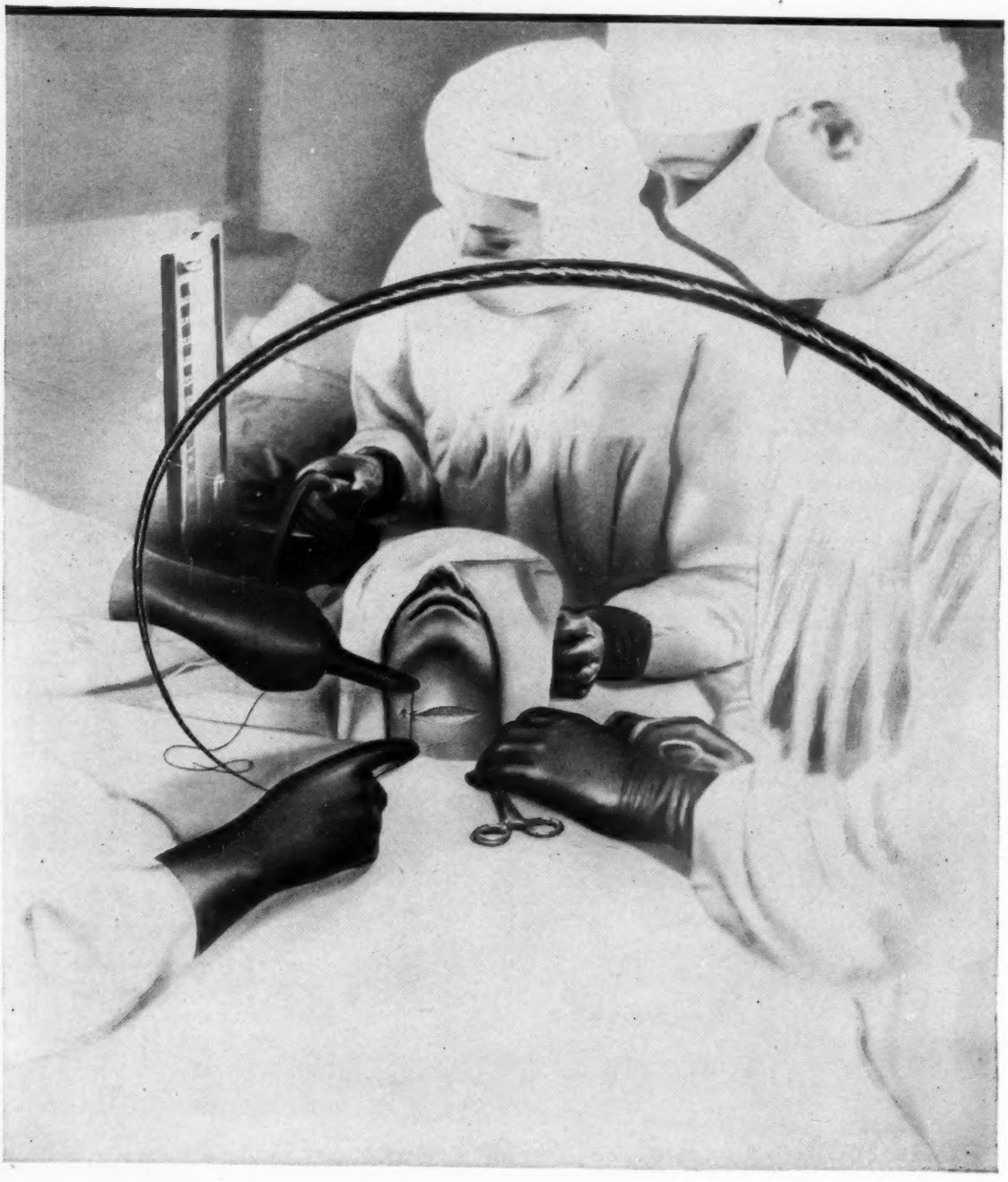

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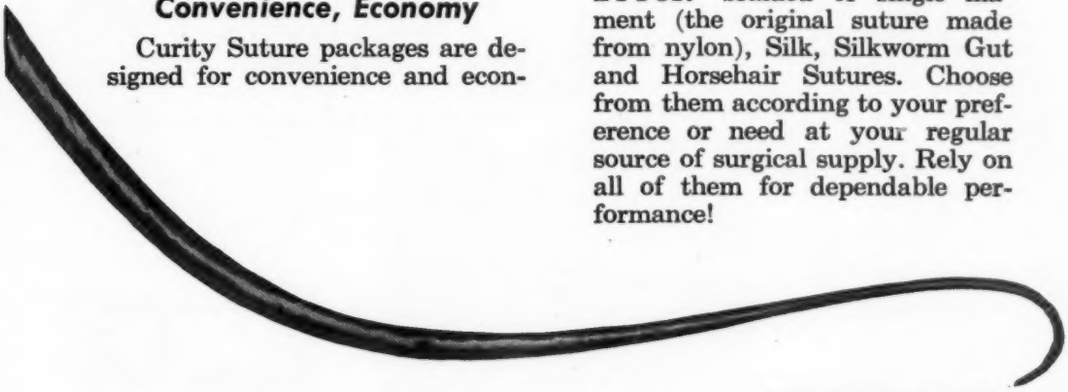

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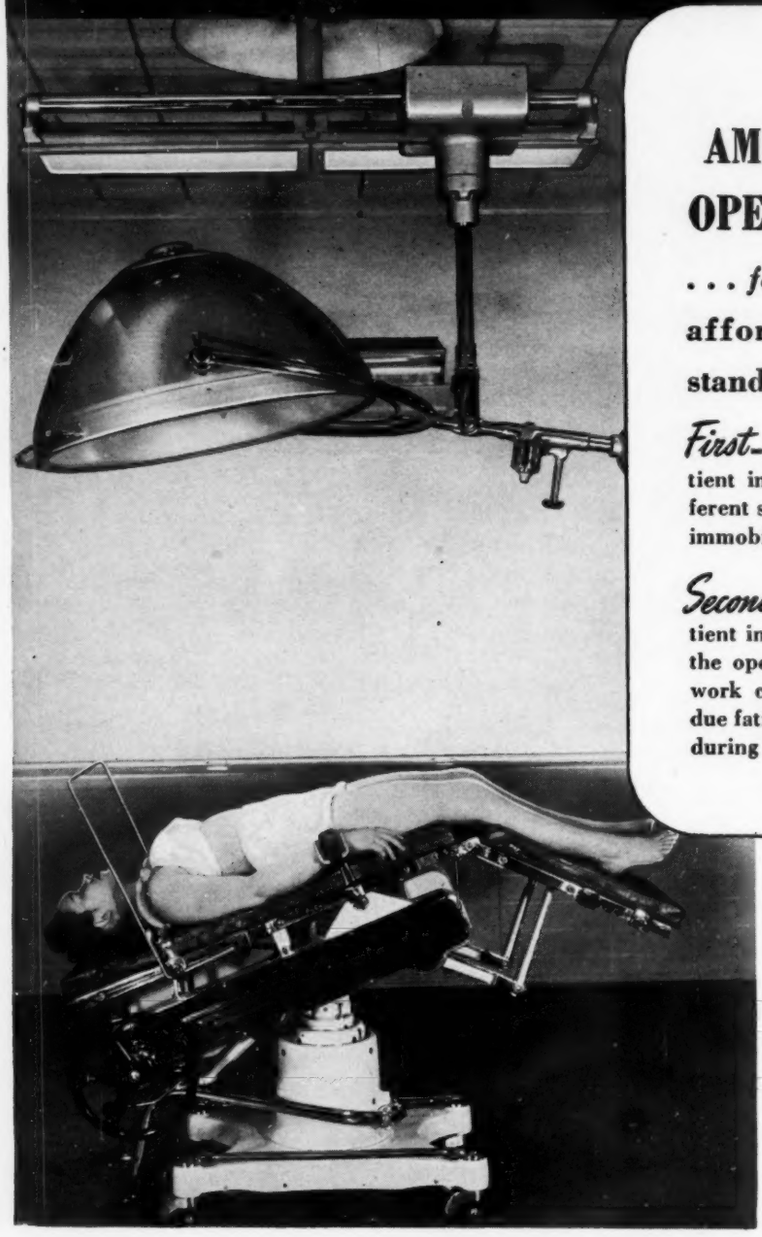

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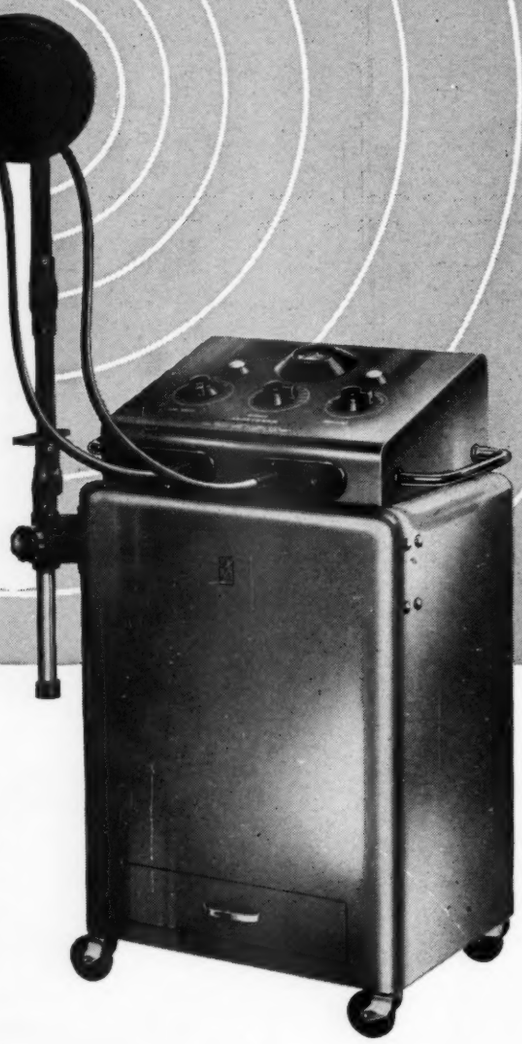

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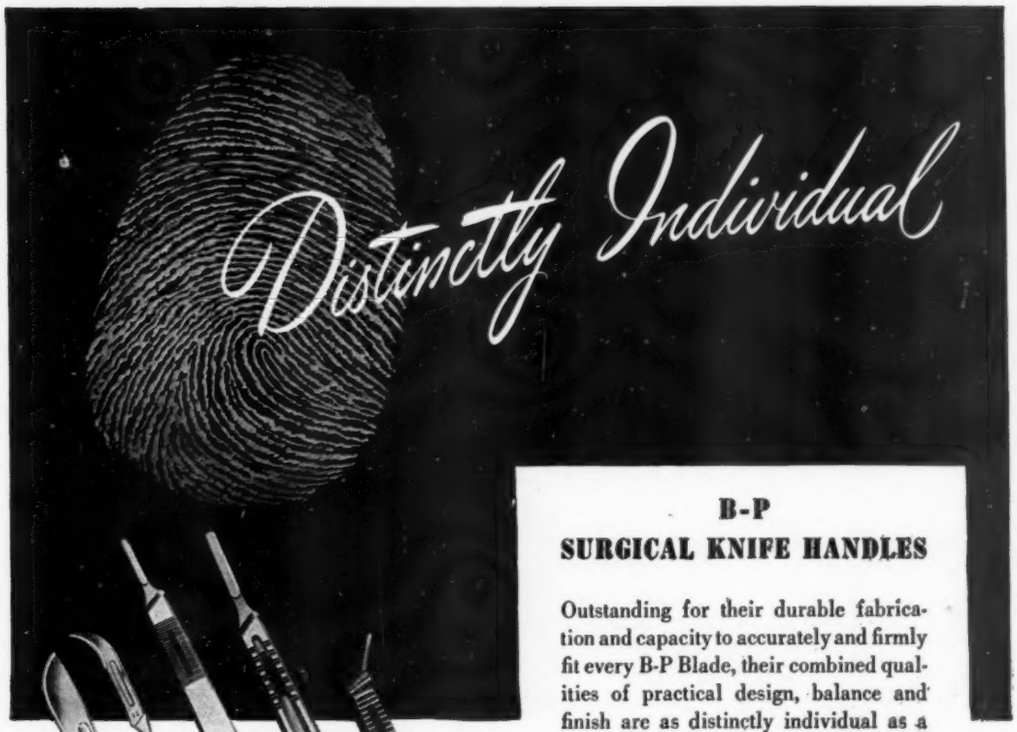

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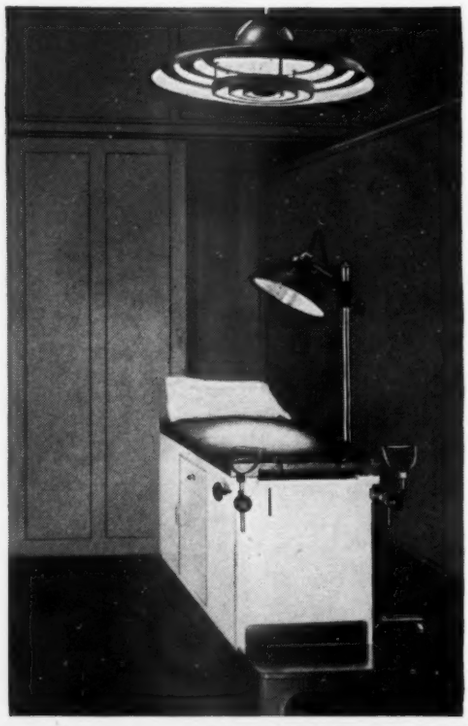

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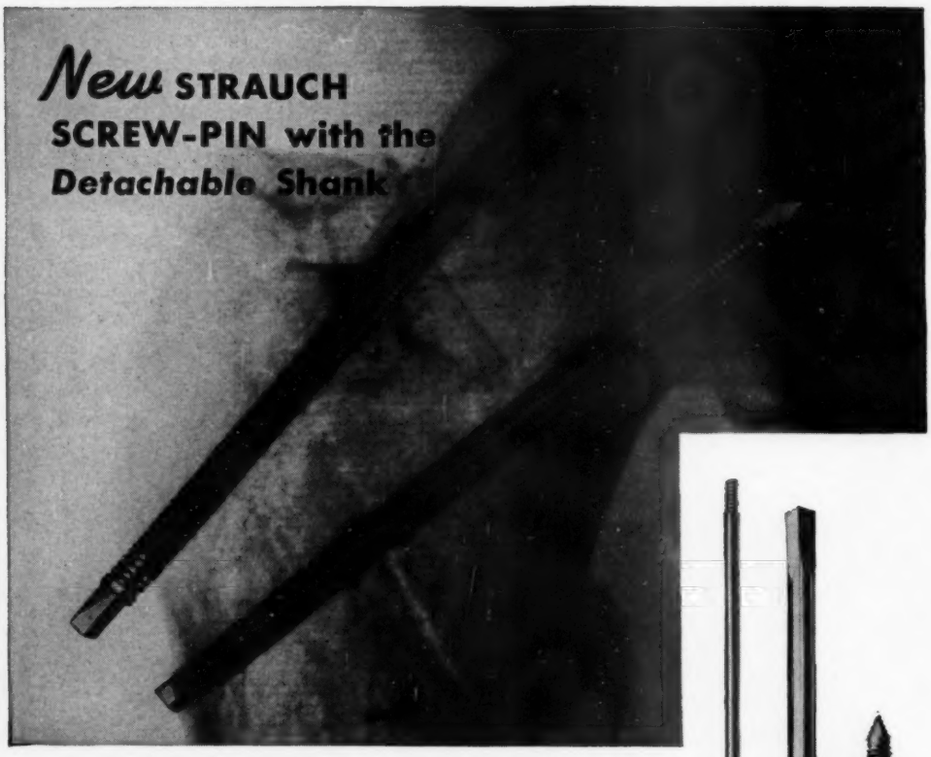

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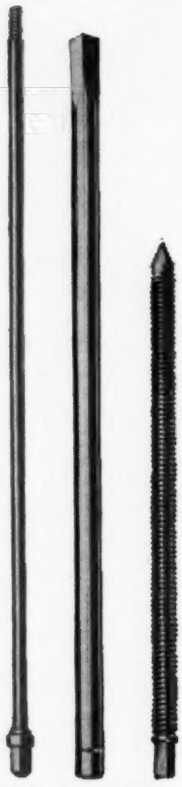

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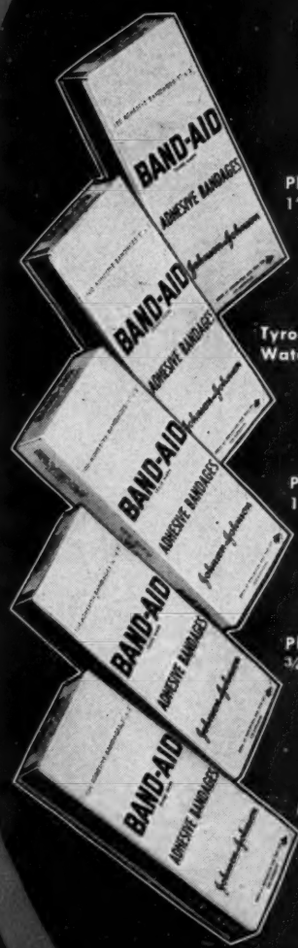

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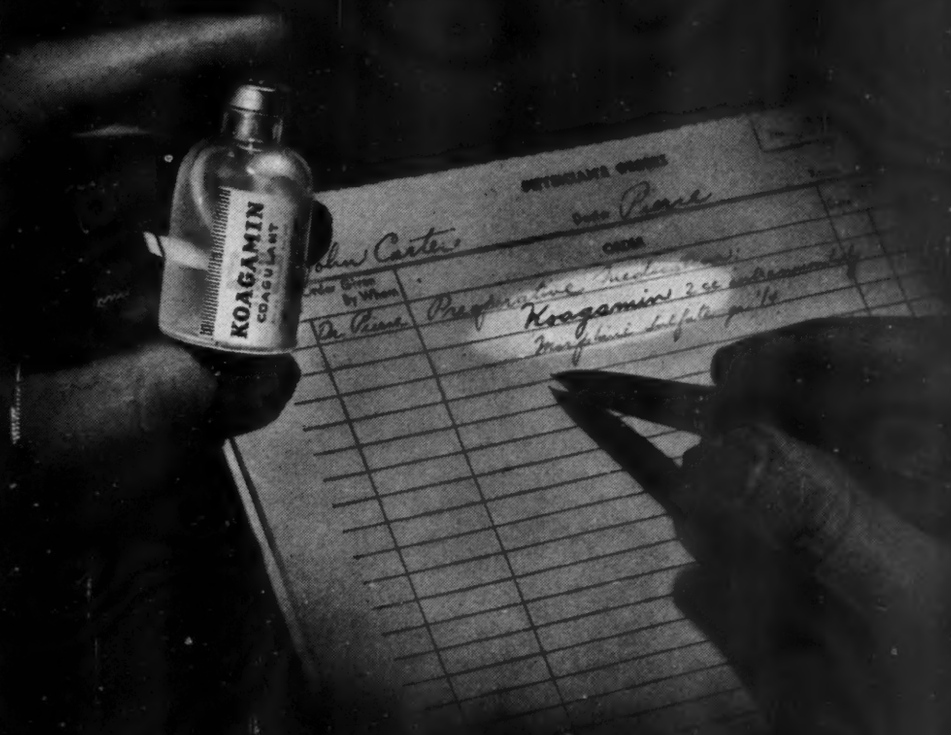

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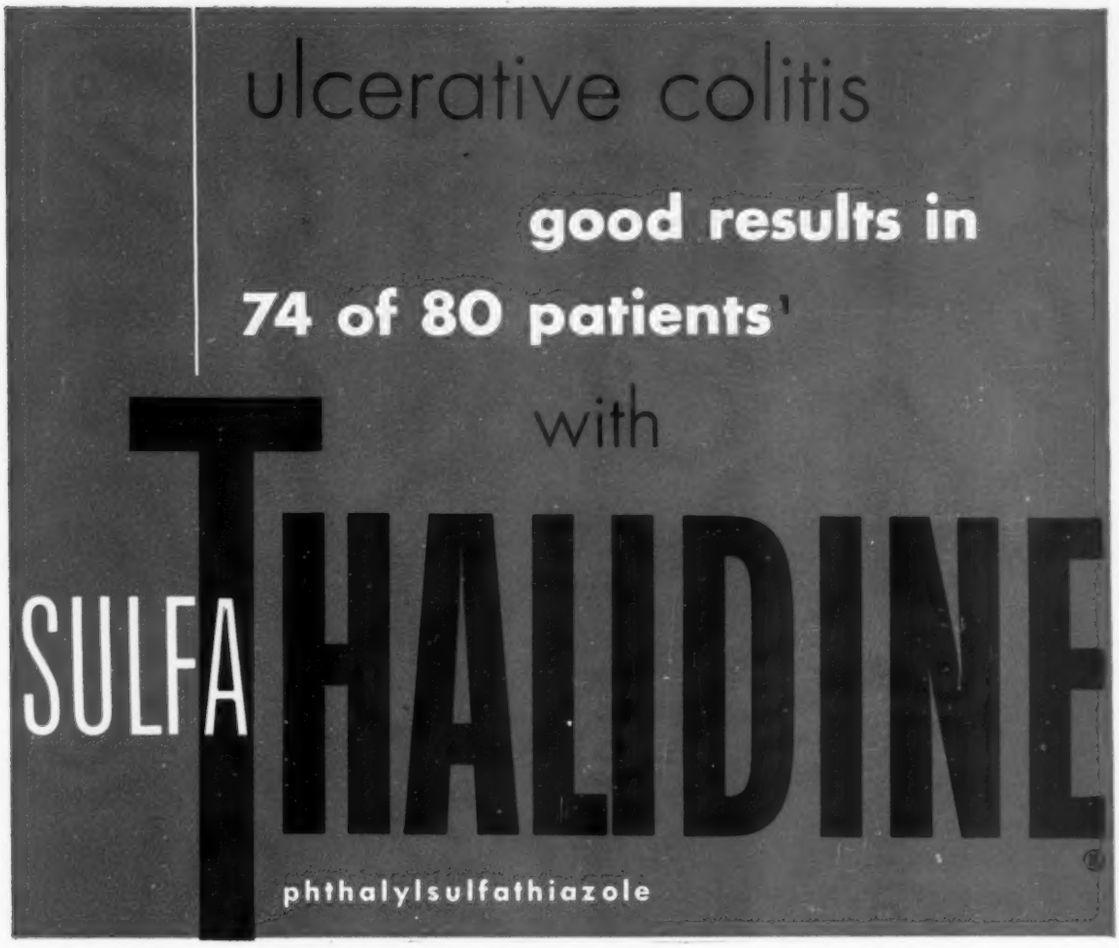

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1. J.A.M.A. 129:1080, Dec. 15, 1945. 2. Illinois M.J. 88:85, Aug., 1945.

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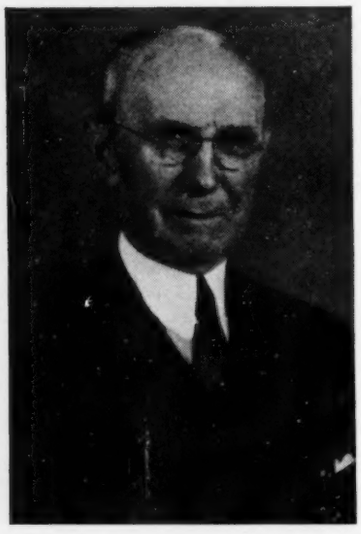

FAIRLEIGH S. DICKINSON, who died June 23, 1948, was born in the town of Beaufort, Carteret County, N. C., the son of David Owen Dickinson and Margaret Anne (Tillman) Dickinson. He was married to Grace Bancroft Smith on December 20, 1916, and had one child, Fairleigh Stanton Dickinson, Jr.

Colonel Dickinson started his career as a seaman for two years on a square rigger, after which he located in Elizabeth, N. J., working for the Singer Sewing Machine Co. The Colonel did not have a formal education but pursued his studies at night during this period. Later he worked with the Saugerties Mfg. Co., Saugerties, N. Y.

In 1896 the Colonel met Mr. M. W. Becton and in the following year, 1897, founded Becton, Dickinson & Company, which was originally located in New York City but later moved to the present location, Rutherford, N. J. The Colonel was president of the company. He was also president of the Rutherford National Bank since 1927.

In 1926 the Colonel was appointed a member of the War Department Business Council. On appointment he was advanced from Lt. Colonel to Colonel. In 1940 he was appointed chairman of the Medical & Surgical Instrument Committee for Medical Departments of the U. S. Army and Navy, and was reappointed to this chairmanship in 1944 by Major General Kirk, Surgeon General of the Army. In 1943 he was appointed by Under Secretary of War Patterson to attend a conference of industrial leaders as the representative of the Surgical Instrument Industry.

YOU will want to see the new *March of Time* film, "The Case of Mrs. Conrad," a story of today's streamlined operative procedure if for no other reason than to keep abreast of what the public will soon be expecting in preoperative and postoperative care as outlined in this movie. Some of the new aspects of modern medical procedure shown in the film are hospital plan benefits; preoperation rest; exhaustive physical examinations and a searching medical history; blood tests and analyses; use of the photoelectric colorimeter; a check for the blood group; urinalysis; basal metabolism test; electrocardiograph; x-rays and the postoperative procedure of early ambulation.

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(*continued on page* 38)

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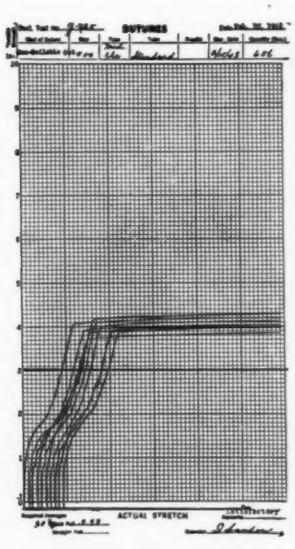

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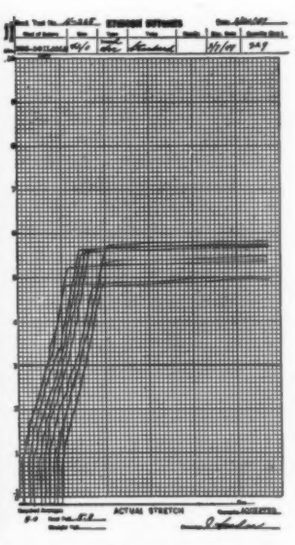

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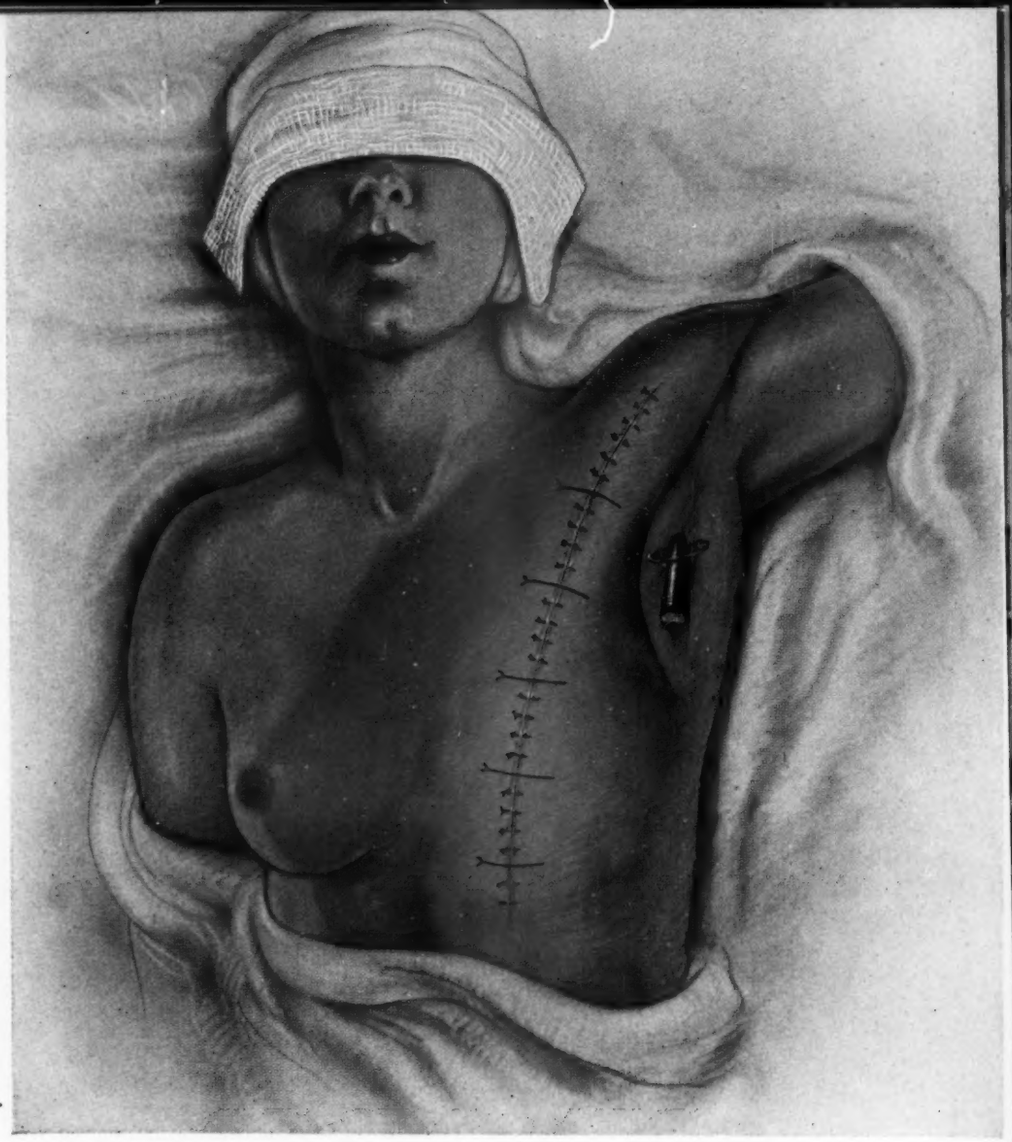

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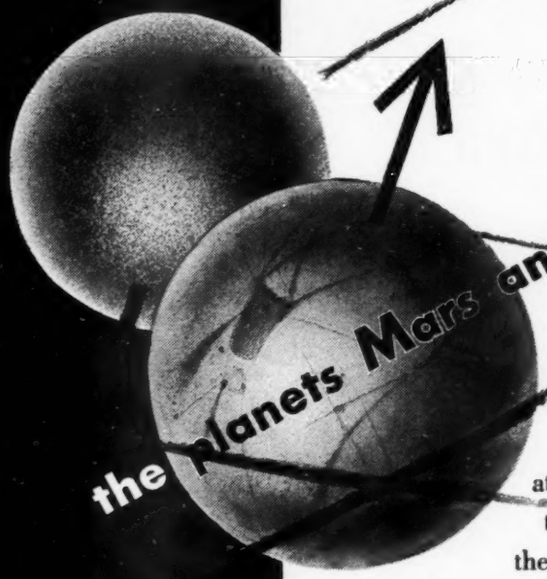

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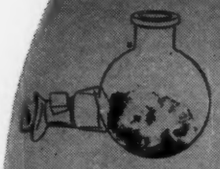

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*Tuohy, E. B.: Practical concepts in anesthesia, Ohio State M. J., 43: 148 (Feb.) 1947.

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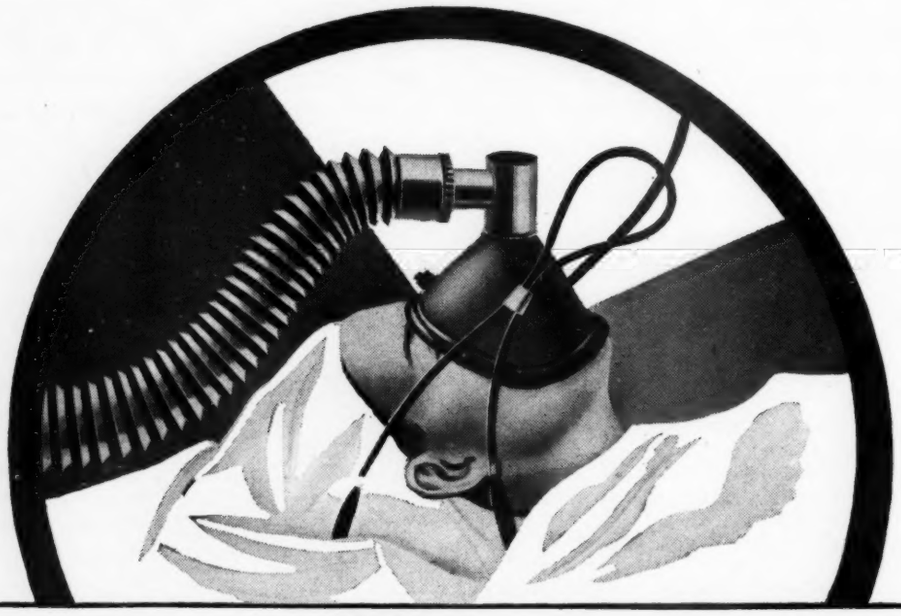

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Fifty-seventh Year of Publication

VOL. LXXVI AUGUST, 1948 NUMBER TWO

Editorial

THE "SILENT" GALLSTONE

IN cases in which gallstones are associated with symptoms of reasonable severity, doctors on the whole have no hesitation in strongly advocating that operation be undertaken for their removal since this is the only way to achieve that object. On the other hand, when these stones are so silent as to scarcely obtrude their presence upon the feelings of their host a very different attitude has been, and still is, widely adopted. This is one of *laissez faire*, exemplified by the patient who triumphantly tells of the advice once given her in some such terms as "My doctor told me that so long as my gallstones don't trouble me I shouldn't trouble them." Since such advice can only have its inception in a lack of knowledge or appreciation of the grave potentialities of stones in the biliary tract, it was believed that a brief discussion of the subject, prompted as it is by certain recent experiences of the writer, would not be inopportune.

While everyone does, or at least should, know that neglect of biliary tract disease often leads to complications of a serious and sometimes fatal nature, we believe that what is not so well known is the corollary, obviously of the greatest importance, that in biliary tract disease, complications arise almost entirely in cases in which gallstones are present and practically never in those in which they are absent. There are two reasons for this: The first lies in the marked propensity which gallstones have for causing obstruction, partial or complete, of some portion of the biliary tract, the result of their presence in a hollow muscular system of variable caliber in which they are able to move about. Secondly, inflammation, acute or chronic, of at least part of the tract is a frequent accompaniment of gallstones. Hence the complications which may, and frequently do, arise in such cases may be due to (1) obstruction, (2) inflammation or (3) to a combination of both these factors, this combination having more baneful effects than either of its component elements acting alone. Thus, within the orbit of complications are to be found multitudinous conditions which include cholecystitis, acute and chronic, hydrops and empyema of the gallbladder, pancreatitis, acute and chronic, obstructive jaundice, cholangitis, acute and chronic, internal biliary fistula, etc. Some constitute an acute threat to the life of the afflicted individual, others act in a no less certain but more chronic and insidious manner by damaging liver function. In the former type the acute nature of the symptoms usually assures that medical advice will be quickly sought, and it is found that this type, although serious enough, fortunately often occurs sufficiently early in the progress of the disease

of the biliary tract to enable early diagnosis and proper surgical treatment to result in a large percentage of cures. The tragedy of the latter type lies in the fact that mildness or absence of symptoms may for some time engender a false sense of security, obscuring the necessity for surgery which alone can hold any hope of cure and so by the time symptoms have become severe enough to force the issue, it may already be too late to combat the pathologic changes which have occurred consequent upon the unchecked progress of the disease. An example of this type of case was a middle-aged woman who had had symptoms suggestive of biliary tract disease for some years but did not seek medical advice until impelled to do so by the symptoms' increased severity during the previous two months. At operation there was found a small pericholecystic abscess containing thick, foul-smelling pus and a single faceted gallstone; thick, vascular adhesions and the poor condition of the patient precluded further exploration. Autopsy revealed a cholecystoduodenal fistula, several gallstones, suppurative cholangitis and multiple liver abscesses. Such a case as this shows us only too clearly the penalty which may have to be paid by the possessor of gallstones if they are allowed to remain until they can indulge their habit of initiating progressive pathologic changes in the biliary tract. Since it is not possible to know just when they may begin to do so, it is obvious that the sooner their removal is undertaken the better the chance of preventing the occurrence of complications.

No discussion of the complications associated with gallstones would be complete without mention of carcinoma of the gallgladder. In the first place, it is recognized that, except in rare instances, this condition occurs only in gallbladders in which stones are present and more particularly when these are of the larger variety and few in number—the very type so often associated with few or no symptoms. This fact is of importance as it leaves us no choice but to admit that the presence of stones in a gallbladder can act as a factor predisposing this viscus to the development of carcinoma. This being so, while it is fortunate that this disease is relatively uncommon, there is still no way of knowing in which cases it will or will not occur if the stone-containing gallbladder is allowed to remain *in situ* and, in addition, it should always be borne in mind that carcinoma is no respecter of persons. Further, it cannot be too strongly emphasized that nearly every case of carcinoma of the gallbladder when explored surgically proves to be hopelessly inoperable so that for all practical purposes it can be said to be a uniformly fatal disease. It follows from this observation that the only treatment which holds any hope is essentially one of prophylaxis and consists of removal of the stone-containing gallbladder before it has become carcinomatous. Yet in one of the writer's recent cases the medical advice given to the patient when gallstones, three in number and of large size, were first demonstrated by roentgenography three years previously was that there would be time enough to deal with them if and when they began to cause troublesome symptoms. Although these had been present only between two and three months when operation was performed, and even then could not be described as severe, carcinoma of the gallbladder was found with widespread peritoneal carcinomatosis and a liver already the seat of metastasis. One can reasonably assume that carcinoma was not present at the time the gallstones were discovered so that in this case the sense of security resulting from the absence of symptoms, added to a disregard of the warning their presence alone should have conveyed, led to neglect in taking timely advantage of the opportunity that presented itself for surgical treatment which alone could have saved the patient from this fatal complication.

What can be done to prevent the occurrence of such tragedies? We shall always have with us the individual who shuns the search for medical advice until it is too late, or he who refuses to run the low risk of the

operation which will restore him to health; such individuals are acting within their personal rights and little, if anything, can be done to help them. However, the tragedies, the prevention of which this communication is concerned, are those which occur in patients as a result of fatal complications which have arisen because of delay in treatment that is the direct outcome of advice given by accredited members of the medical profession. We therefore wish to emphasize first, that because gallstones are an important causal factor in the spread of pathologic changes in the biliary tract they constitute a definite menace to the future health of the individual who harbours them; and secondly, that when gallstones are present, absence of symptoms is no guarantor of safety for serious complications may be in, or have actually progressed beyond, their incipient stage before symptoms of any severity manifest themselves. It is evident, therefore, that prevention of complications means not only the removal of gallstones and the concomitant treatment of any related pathologic condition, but that the operation necessary to achieve these objects should be carried out as soon as possible after either the presence of gallstones has been demonstrated or the diagnosis of biliary tract disease has been made. The only cases which should be considered as qualifying for exception to this rule are those in which associated conditions render the operative risk too great. Until physicians are willing to recognize these facts and to impress them upon patients with gallstones who seek their advice, avoidable tragedies will continue to occur.

Let us therefore keep constantly in mind that "Gallstones are a serious possession" (an apothegm of Rutherford Morison's) and in particular not allow silent gallstones to deceive us into regarding their removal as any the less essential than that of the "noisy" variety.

James H. Saint, M.D.

Original Articles

TECHNIC FOR RESECTION OF THE RIGHT MIDDLE AND RIGHT LOWER LOBES OF THE LUNG

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THE mortality and morbidity rates for pulmonary resection are now sufficiently reduced that many diseases of the lungs presenting problems in therapy have has seen the development of advancements in technic; but in addition, this experience has permitted the accumulation of data concerning criteria of operability and post-

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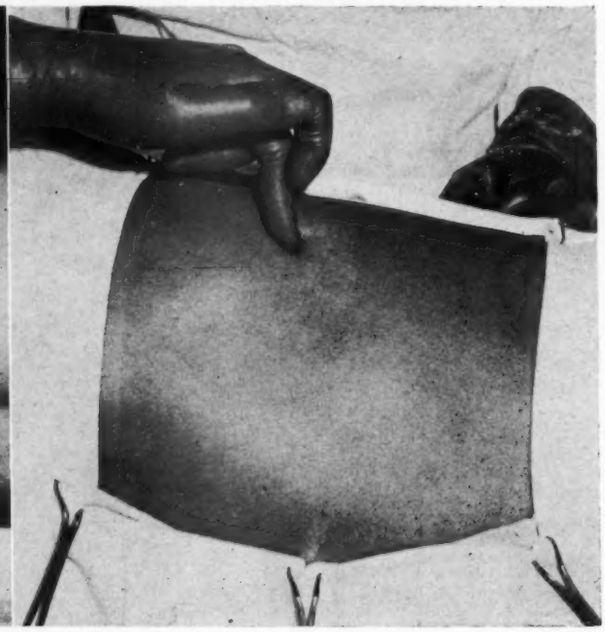

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FIG. 1. Endotracheal anesthesia is in progress; a blood pressure cuff has been applied to the right arm and an intravenous needle has been placed in an antecubital vein of left arm.
FIG. 2. The field painted and draped; finger indicating the inferior angle of the scapula.

come within the range of satisfactory treatment.

Repeated performance of lobectomy has evolved a fairly standardized technic for the operation. This technic is largely concerned with the maneuvers necessary for the isolation and the secure interruption of blood and air trunks supplying the lobes resected.

Increasing experience with lobectomy operative treatment which have contributed much to improved effectiveness in the case management of the patients subjected to lobectomy.

The primary object of this presentation is to describe the technic of resection of the right middle and lower lobes in a clear, brief manner. The surgical technic is then integrated with the other phases of case management by the inclusion of a representa-

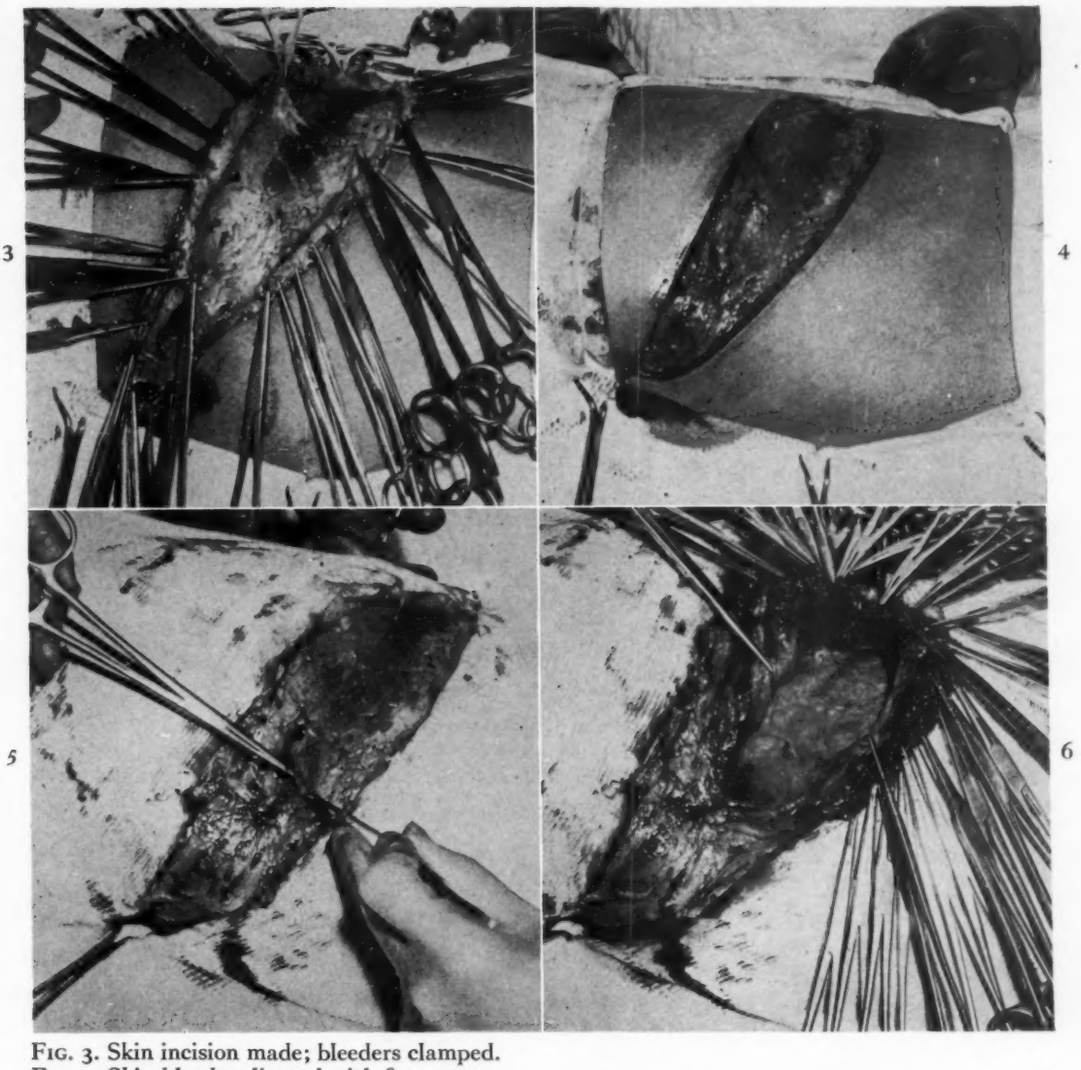

FIG. 3. Skin incision made; bleeders clamped.
FIG. 4. Skin bleeders ligated with fine cotton.
FIG. 5. Cotton stockinette applied to wound edges; fascia of the auscultatory triangle picked up and incised.
FIG. 6. The latissimus dorsi and the serratus anterior muscles cut.

tive case report from diagnosis of the disease to discharge of the patient from the hospital.

Review of 219 non-tuberculous broncho-pulmonary lesions found in the diagnostic studies carried out in this endoscopic clinic immediately prior to the preparation of this presentation, and constituting a random check, showed that in ten cases the pathological condition was limited to the right middle and lower lobes alone. Right middle and lower lobe lesions occurred with varying degrees of involvement on the contralateral side in twenty other cases in the same series. This suggests that lobectomy of the right middle and lower lobes might be considered in the treatment of approximately 14 per cent of the entire series.

Tabulation of the last one hundred lobectomies performed (segmental resection, except lingulectomy, excluded) shows lobectomy of the right middle and lower lobes was carried out in ten cases. In the same series of cases the right middle lobe alone was removed nine times and the right lower lobe was removed alone in thirteen caes. This series included cases of pulmonary tuberculosis in which one lobe or more was

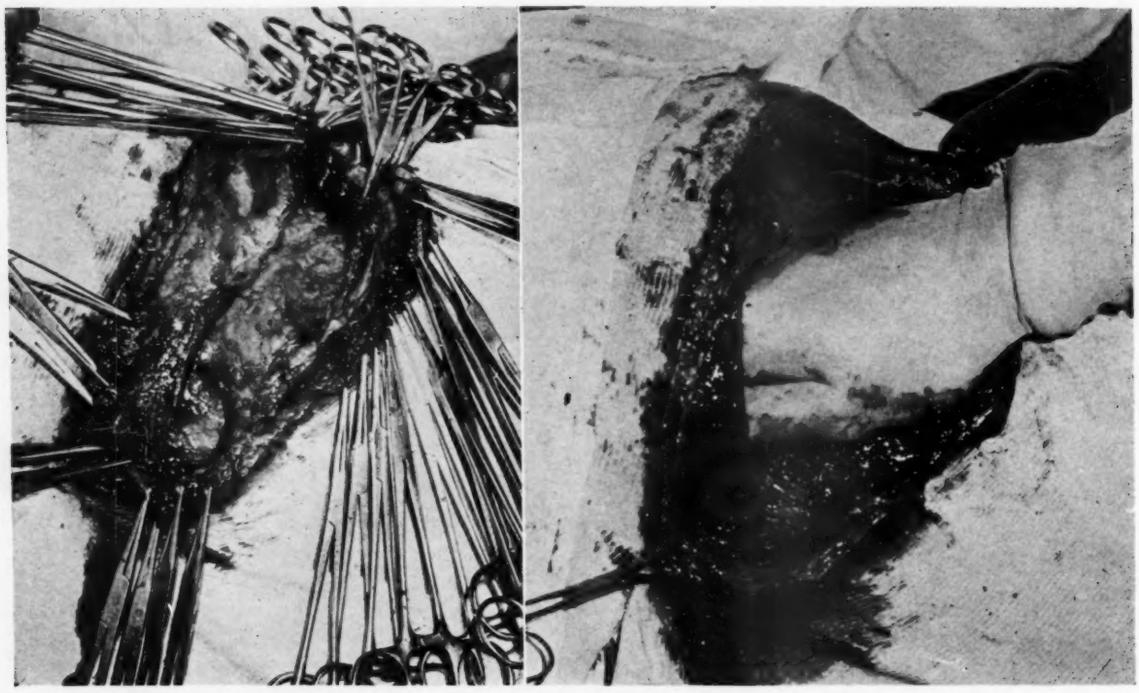

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Fig. 7. The muscle incision extended posteriorly with division of the exposed portions of the trapezius and rhomboid major muscles; muscle bleeders ligated with fine cotton.
Fig. 8. The right hand passed under the scapula to palpate the prominent attachment of serratus anterior on the second rib, and the first rib above it.

removed; segmental resections for tuberculomas were excluded.

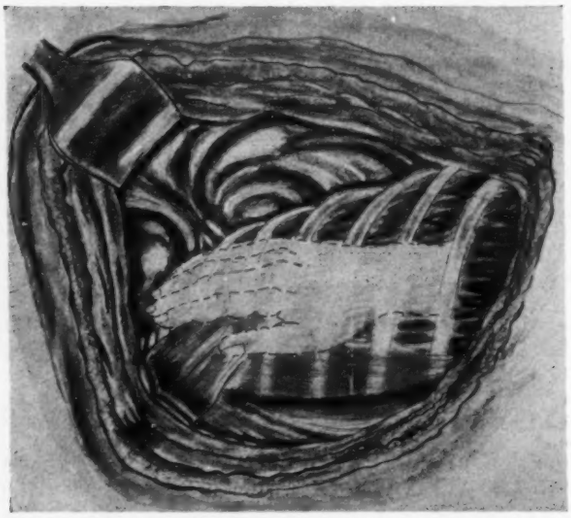

Fig. 9. Diagram showing the serratus anterior attachment to the second rib. The first and second ribs are usually identifiable by palpation.

The totals suggest that the technic of excision of the right middle or the right lower lobe, or both, will have direct application in approximately one-third of all cases in which lobectomy will be done.

Teamwork among all concerned with the management of the case, from endoscopist to physical therapist, has demonstrably influenced the results obtained in lobectomy. The anesthetist may merit special mention, for the operation should rarely if ever be attempted without the services of an anesthetist who is geared by training, equipment and temperament to the exigencies of the operation.

TECHNIC

Incision. The skin incision for lobectomy of the right middle and lower lobes is made in a manner which will permit partial mobilization of the scapula and exposure of the sixth rib. With anesthesia induced and respiration controlled by tracheal intubation, the patient is positioned on the left side. (Fig. 1.) In this position the inferior angle of the scapula overlies the sixth rib. (Fig. 2.)

The skin incision extends obliquely an-

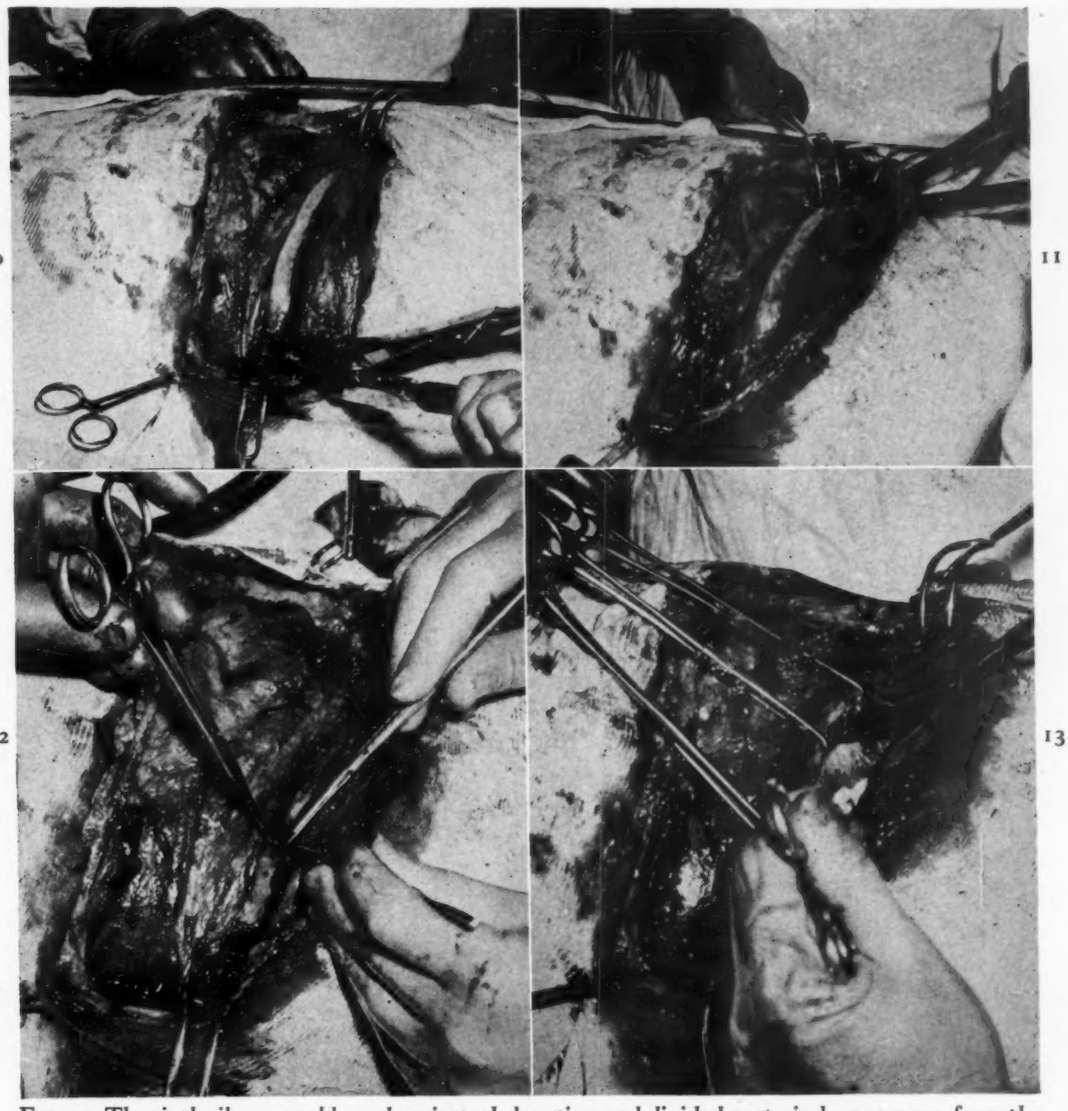

FIG. 10. The sixth rib exposed by subperiosteal elevation and divided posteriorly 2 or 3 cm. from the angle.
FIG. 11. The Bethune rib shears divide the sixth rib anteriorly.
FIG. 12. The periosteum and parietal pleura in the bed of the sixth rib picked up and incised.
FIG. 13. Incision in the pleura completed for the length of the resected portion of the sixth rib; adhesions between the lung and chest wall divided, using a "dissector" at this stage.

teriorly and downward over the course of the sixth rib from a point 5 cm. from the dorsal vertebral spine to the posterior axiliary line, passing inferior to the angle of the scapula.

The incision first includes the full thickness of the skin and the subcutaneous fat to the muscle layer. Bleeders are clamped and tied with fine cotton, No. 50 black. (Figs. 3 and 4.) Skin drapes of stockinette are secured to the skin edges by towel clips applied from the underside of the drapes.

The wound, which is thus completely protected from the skin, is now deepened to the thoracic cage. Incision is made through the auscultatory triangle formed by the converging margins of the major rhomboid, the trapezius and the latissimus dorsi muscles. (Fig. 5.) The finger introduced through the opening palpates the ribs and intercostal structures.

The muscle layer is cut over the finger which advances in the easily separated line of cleavage anteriorly (Fig. 6) and then

Fig. 14. Right stem bronchus exposed above the level of the middle lobe bronchus.

posteriorly and superiorly. (Fig. 7.) Bleeders are caught as they are cut and tied with fine cotton.

Alexander refers to the method of Carter in making the incision in the muscles with reduced blood loss. As the incision is made the muscle on either side is compressed between the finger and thumb of the operator on one side and of an assistant on the other. A momentary release of the compression shows the location of the bleeders and they are at once clamped.

Pleura Opened. The hand sweeps upward and forward beneath the scapula. (Fig. 8.) The attachment of the serratus anterior to the second rib is prominent and provides an easily palpable landmark. (Fig. 9.)

With a scalpel an incision along the center of the sixth rib divides the periosteum over the outer surface of the rib. A Matson or similar type raspatory strips the periosteum free, and a sufficient length of rib is removed with a rib shears to provide exposure when the pleura is opened. A section of rib from the transverse process to the posterior axillary line will usually allow such exposure (Figs. 10 and 11), but further lengths of rib or protruding stumps may be cleared of periosteum with a Sanderson periosteal elevator and cut with a Sauerbruch double action rongeur. About 18 cm. of rib is removed.

The pleura is opened by an incision

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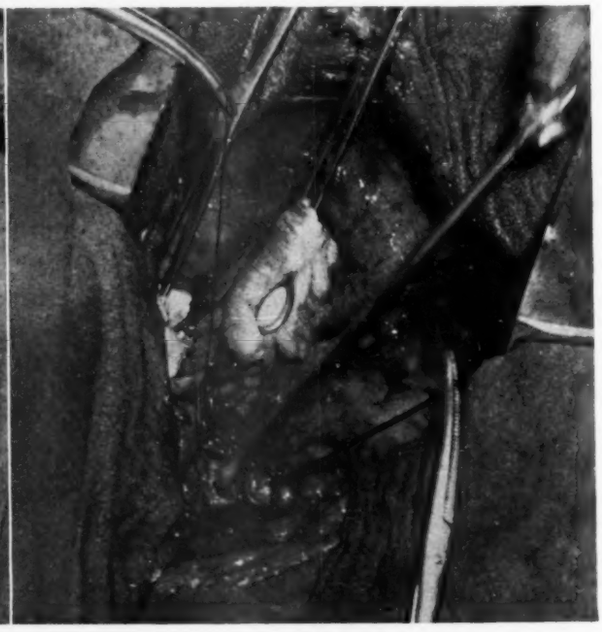

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Fig. 15. Right stem bronchus tied off below; stay sutures inserted above the contemplated level for cutting.
Fig. 16. The bronchus cut and an aspirating unit removes secretion from the proximal stump of the bronchus.

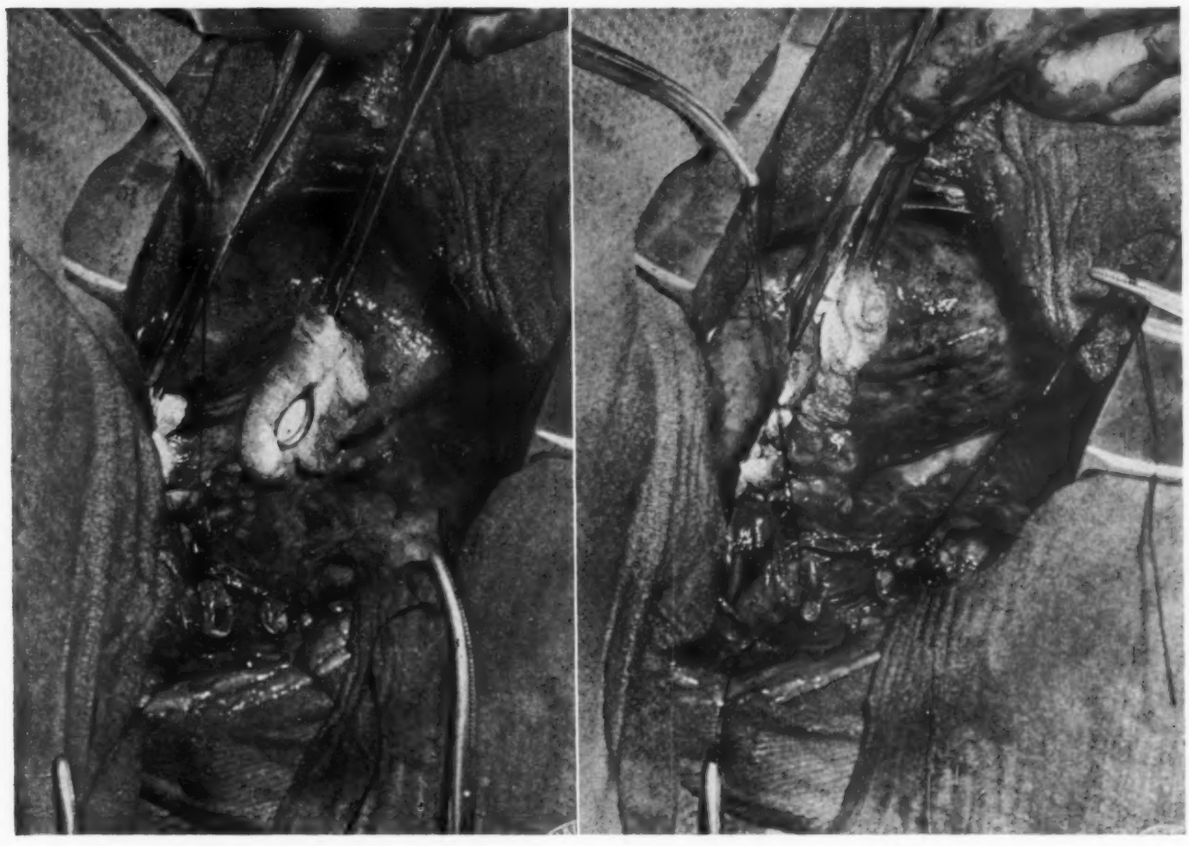

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Fig. 17. The bronchial stump ready for suturing.
Fig. 18. The bronchus closed by a row of interrupted fine No. 00 chromic catgut on atraumatic needle.

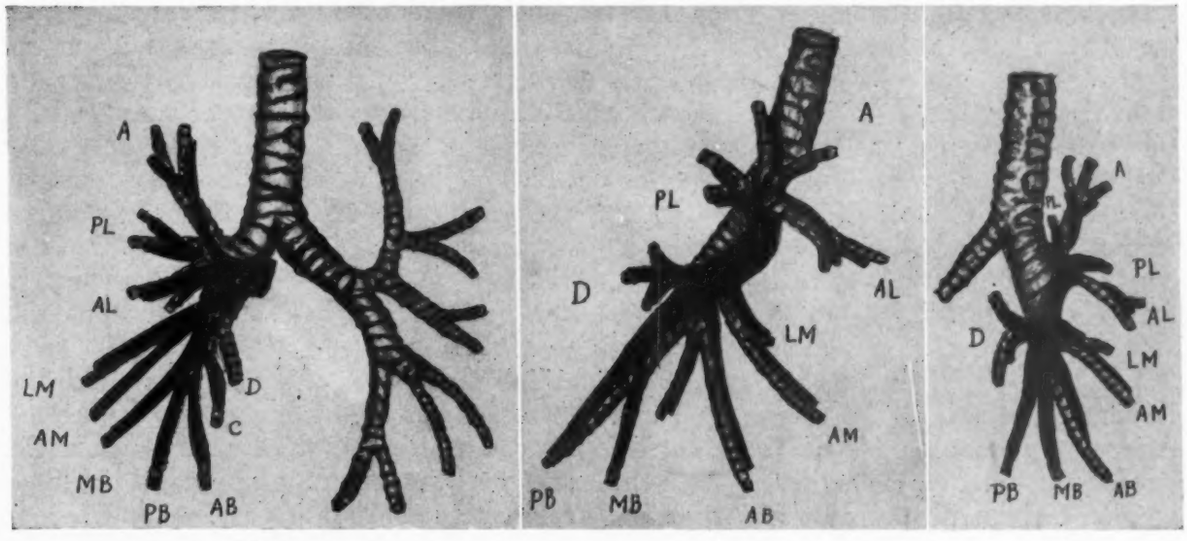

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Fig. 19. Diagrammatic anteroposterior view to show relationship of pulmonary artery to right bronchial tree. The letters indicate the division bronchi of each lobe with the nomenclature used by Foster-Clark. In the right upper lobe "A" indicates apical division; "PL," posterolateral division; "AL," anterolateral division. The relationships shown are based on the studies of Boyden.
Fig. 20. Diagrammatic lateral view. "AM" indicates anterior middle division; "LM," lateral middle division.
Fig. 21. Diagrammatic oblique view from the right and posterior. In the right lower lobe "AB" indicates anterior basic division; "MB," middle basic division; "PB," posterior basic division; "D," the dorsal division of the apical segment of the right lower lobe.

Fig. 22. Pulmonary artery exposed and ligated.

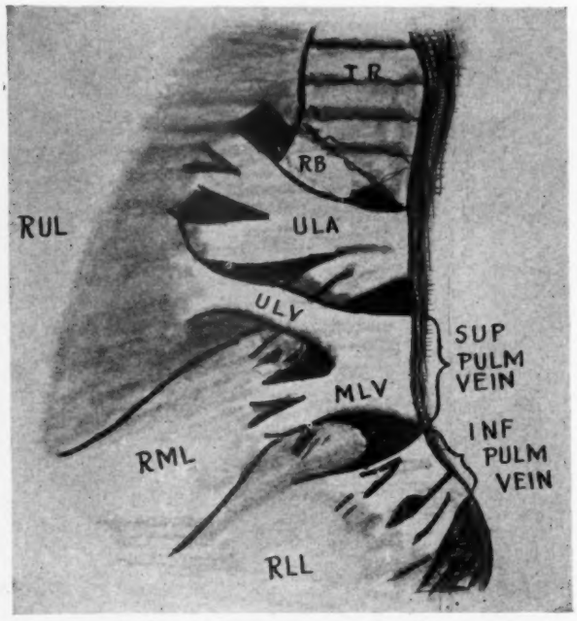

Fig. 23. Cadaver dissection of right hilar region by Kent and Blades. This diagrammatic anterior view shows the middle lobe vein as a tributary of the superior pulmonary vein lying anterior to the pulmonary artery and its branches.

through the periosteal bed of the rib with care to avoid injury to the intercostal structures and to avoid perforation of the lung through an underlying adhesion. (Fig. 12.) The anesthetist is warned as the pleura is about to be opened, so that the respirations of the patient will continue smoothly under control by the anesthetist if necessary.

Pneumonolysis. A complete pneumonolysis, including development of the fissures, is now performed if necessary. (Fig. 13.) This is done by blunt and sharp dissection, and useful instruments in effecting this stage of the procedure are: stick sponges, "dissectors," a long handled scissors of the Reinhoff type and a transluminating lamp. After the pneumonolysis has been carried out for a distance of several inches about the incision in the pleura, the rib spreading retractors are inserted to provide further exposure. The large, heavy, Finochietto rib retractor gives satisfactory exposure; where a smaller type of rib retractor is used, two may be necessary, one in the anterior and one in the posterior angles of the wound. The full thickness of the wound edge on either side is protected by a laparotomy sponge before the rib retractor is applied.

Bronchus Divided. The anesthetist allows a partial collapse of the right lung which is retracted anteriorly with stick sponges. The posterior reflection of the pleura over the hilum is divided; and the bronchus, which may be identified by the palpable cartilaginous rings, is isolated between the orifices of the upper lobe and the middle lobe bronchi. Stripping of the bronchus proximal to the contemplated site of division is avoided. The bronchus is steadied with a large kidney pedicle forceps. (Fig. 14.)

The correctness of the level at which the bronchus has been exposed may be verified by compression of the bronchus while the anesthetist attempts to re-expand the lung. A selective re-expansion of the upper lobe is confirmatory. A sufficient margin of right stem bronchus is allowed to remain to permit closure without obstruction to the upper lobe bronchus. Stay sutures are inserted to control the proximal stump of the bronchus, a distal tie is made and the bron-

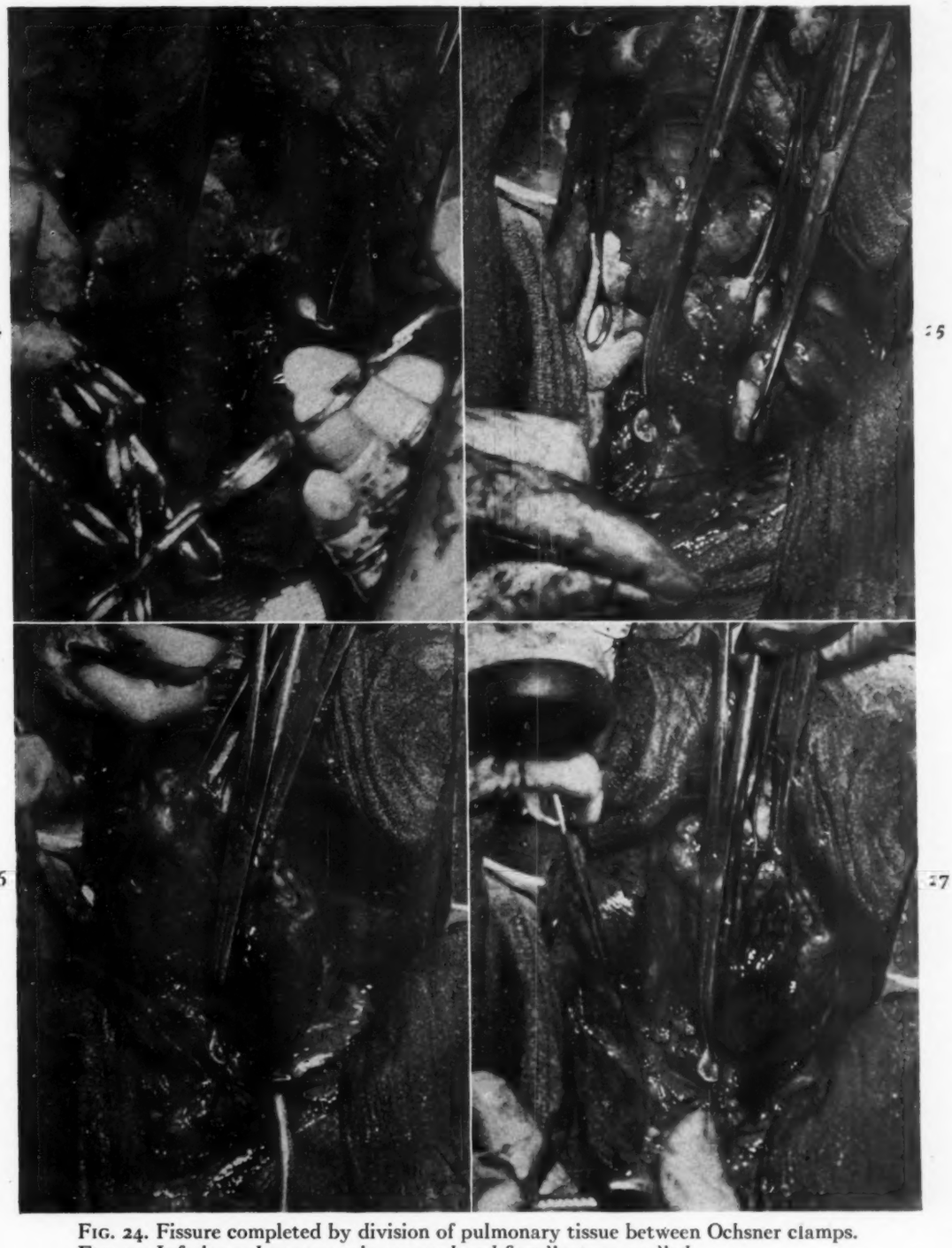

Fig. 24. Fissure completed by division of pulmonary tissue between Ochsner clamps.
Fig. 25. Inferior pulmonary vein exposed and first ligature applied.
Fig. 26. The second and third ligatures applied to the inferior pulmonary vein.
Fig. 27. Pulmonary vein divided leaving two ties on the mediastinal side.

chus is ready for division. (Fig. 15.) The tie on the distal segment of the bronchus prevents the escape of secretion from the lower lobes, and the secretion present in the proximal stump of the bronchus is quickly removed by aspiration. (Fig. 16.) Bleeding from a bronchial artery may be active when the bronchus is divided, and the artery may require clamping and ligation with quilting cotton.

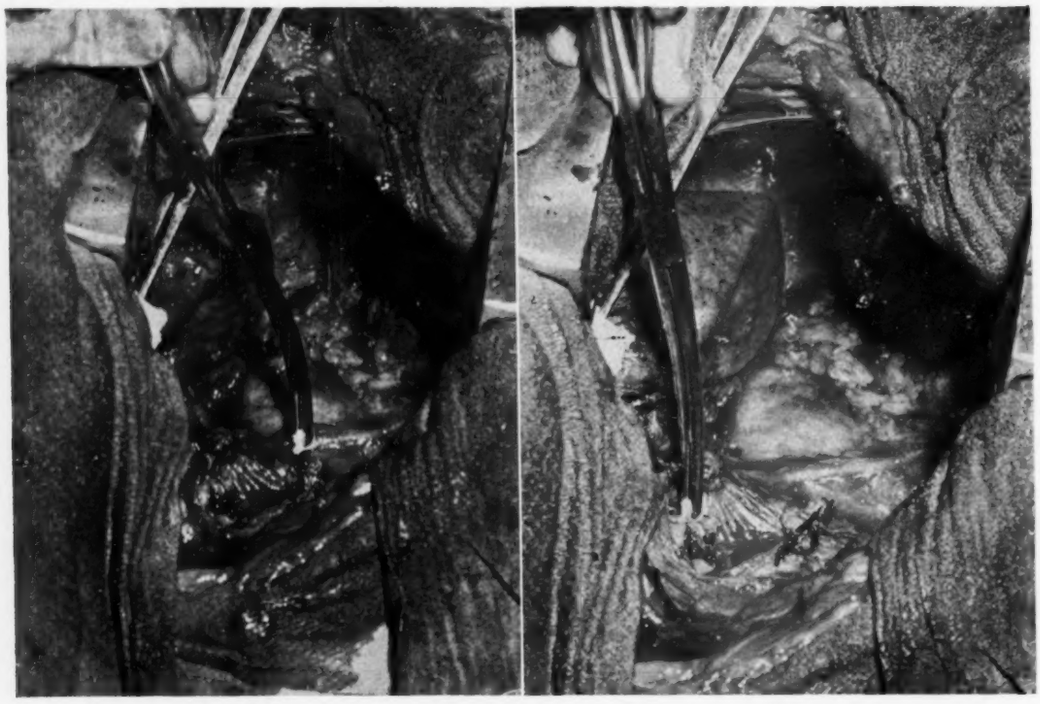

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Fig. 28. The right middle and lower lobes removed. The dissector indicates the ties on the stump of the inferior pulmonary vein.

Fig. 29. The dissector indicates the ties on the pulmonary artery and the sutured bronchial stump. The pleural flap remaining from the incision into the pleura over the hilum lies posteriorly in folds.

Closure of Bronchial Stump. Airtight closure of the stump of the bronchus is effected by four or five interrupted sutures of catgut, No. 00 chromic, on an atraumatic needle. Mattress sutures may be employed, but an effort is made to produce minimal impairment of the bronchial stump circulation. (Figs. 17 and 18.)

Ligation and Division of Pulmonary Artery. The pulmonary artery is next sought and isolated by gentle, blunt dissection, and sufficient of its branches are exposed to insure preservation of the blood supply to the upper lobe. (Figs. 19, 20 and 21.) The dissection of the hilum may be rendered difficult by the presence of enlarged adherent lymph nodes.

The artery is ligated proximal to the branch or branches to the middle lobe by means of a silk tie, after which a similar tie is placed distally allowing an interval of about 1 cm. between. If the artery bifurcates in this interval, the branches may be tied separately. (Fig. 22.) A stick tie is then applied to the artery just distal to the proximal tie, and after this is secured the artery is divided below it leaving a small cuff proximally. No. 18 silk has been used usually for the free ties on the artery and No. 5 silk for the transfixion ligature.

The vein from the middle lobe, usually a tributary of the superior pulmonary vein, lies anterior to the artery (Fig. 23), and it is isolated, tied and divided in a manner similar to that employed for the artery. Anatomical variations in artery and vein patterns are common and care must be used in the identification of the vessels included in the dissection.

The middle lobe is now separated from the upper lobe by division of the intervening pulmonary tissue between Ochsner clamps. (Fig. 24.) The wound in the upper lobe is closed by a double row of continuous fine catgut sutures on an atraumatic needle.

The middle and lower lobes now remain attached to the mediastinum by the inferior pulmonary ligament and the inferior pulmonary vein which it contains. The isolation of

FIG. 30. Pleural flap sutured over bronchial stump.
FIG. 31. Right upper lobe re-expanded by anesthetist.
FIG. 32. Wound reinspected after irrigation.
FIG. 33. Finochietto rib retractor about to be removed from chest; right upper lobe expanded.

the inferior vein is facilitated by an incision in the lower margin of the inferior ligament and gentle dissection upward with the finger introduced through the opening in the ligament. Care is exercised not to produce a tear in the thin wall of the vein as the ligament is divided and the vein is cleared of as much of the adjacent tissue as possible. A No. 18 silk, free tie is applied to the vein at the most proximal point in its exposure. (Fig. 25.) A stick tie of the same material is applied just distal to the first tie (Fig.

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FIG. 34. Rubber angle drains; above as purchased; below as refashioned before use.
FIG. 35. Angle drains; left as purchased; right before use.

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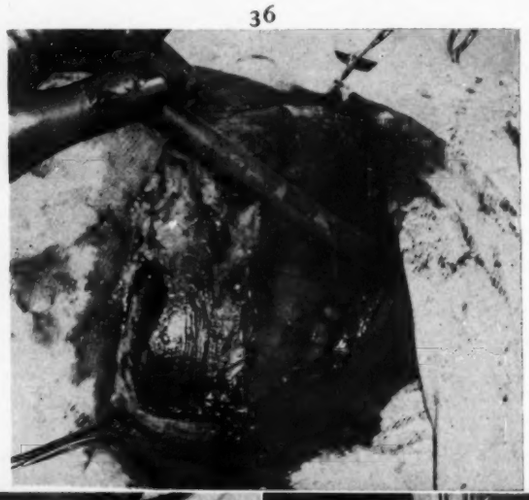

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FIG. 36. Posterior angle drain inserted by withdrawal through stab wound.
FIG. 37. Large hemostat follows points of scissors into chest through anterior stab wound.
FIG. 38. Anterior drain withdrawn through stab wound.

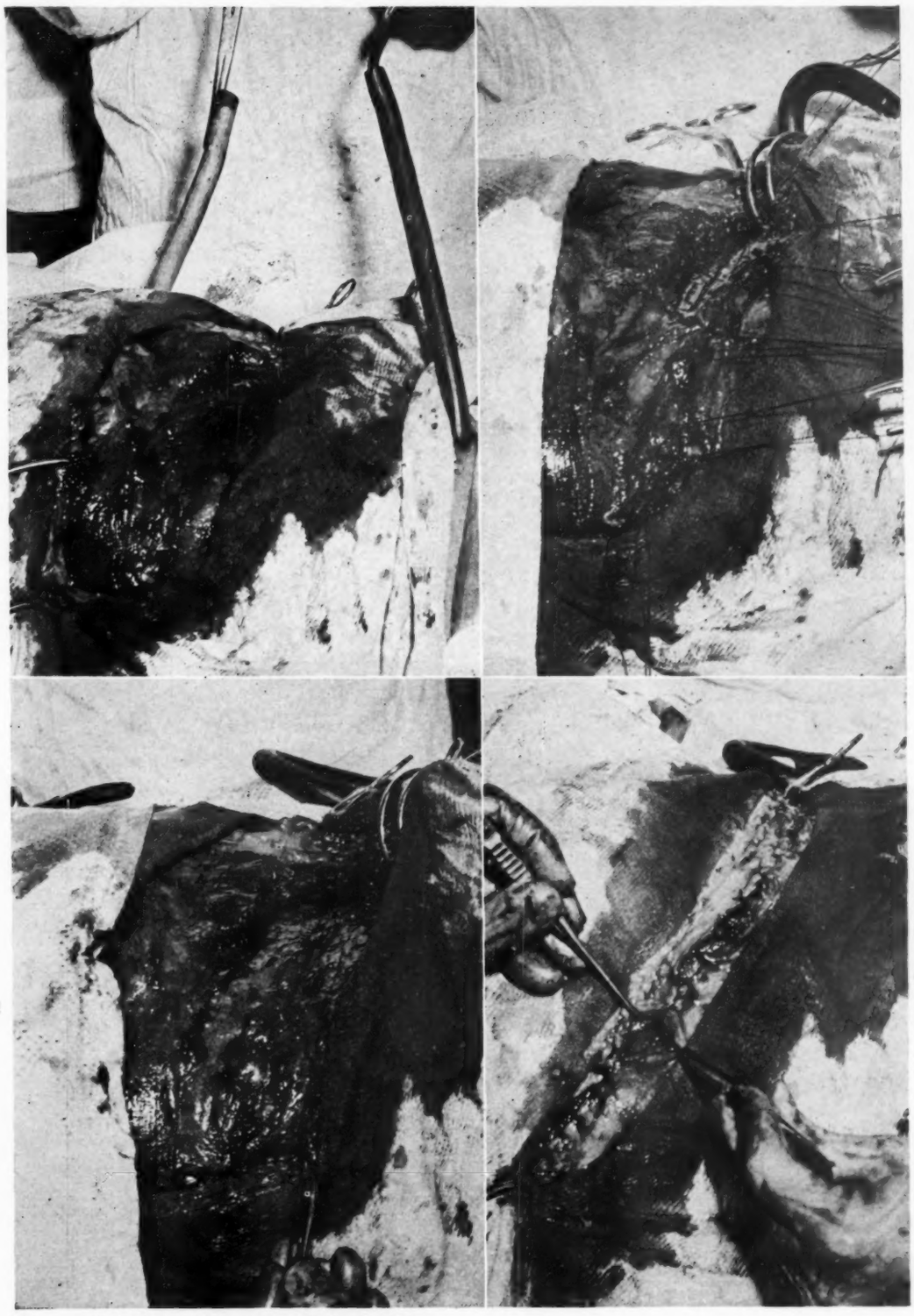

Fig. 39. Anterior and posterior drains in place.
Fig. 40. Heavy pericostal silk suture about fifth and seventh ribs has been drawn up and tied to close wound, while smaller interrupted nylon sutures are tied closing the wound in the rib bed.
Fig. 41. Heavy silk suture cut out and reconstruction of chest wall muscles started.
Fig. 42. Closure of auscultatory triangle.

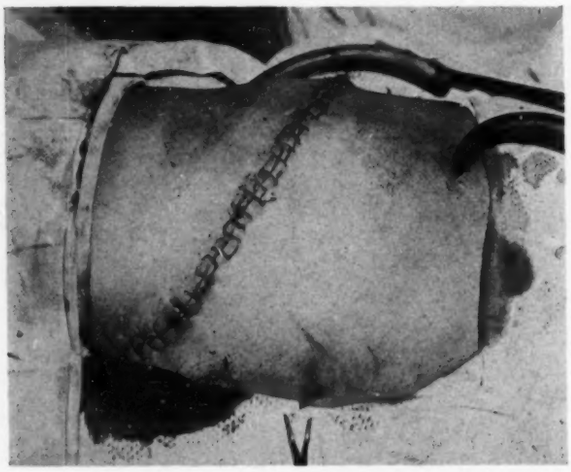

Fig. 43. Wound closure completed; chest drains will be connected to water seal after lung is re-expanded.

26), after which a third tie is made sufficiently distalward to permit division of the vein between ties No. 2 and 3.

The vein is then cut and the lobes are removed from the chest. (Figs. 27 and 28.) (In Figure 28 the tie on the inferior vein is indicated. In Figure 29 the ties on the artery and the closure of the bronchus are pointed out.)

Pleural Graft. The leaf of pleura remaining from the posterior incision of the hilum is brought anterior as a flap to cover the bronchial stump. It is secured in place by several interrupted sutures of fine catgut. (Fig. 30.)

Insertion of Drains. Before the bronchus is covered by the pleural flap, it may be tested for the airtightness of the closure by increase in the pulmonary pressure produced by the anesthetist. With the pleural graft placed, the anesthetist inflates the upper lobe (Fig. 31) and the suture line in the upper lobe is again inspected for adequate closure.

Any blood or secretion which may have escaped into the chest is removed by irrigation with normal saline and aspiration. If the surgeon elects to produce a temporary paralysis of the right diaphragm, it is done at this stage by crushing the phrenic with the points of a hemostat where the nerve courses over the pericardium. A final check for hemostasis (Fig. 32) is made and the rib retractors are removed. (Fig. 33.)

A No. 45 French rubber, angle drain, the tip of which has been cut off and in which a new opening has been cut (Figs. 34 and 35), is now inserted in the posterolateral chest through an intercostal space about 3 cm. above the costophrenic angle.

The skin incision for the drain is of such size that it will fit snugly about the drain when inserted. The incision is made with a scalpel over the tip of a heavy scissors which has been forced outward through the selected intercostal space. A long curved hemostat follows the scissors into the chest through the incision so made, and the drain is drawn out through the opening by the withdrawal of the clamp. (Fig. 36.)

A second drain of the same type is inserted through the fourth intercostal space anteriorly, the wound being placed sufficiently far lateralward to avoid the nipple and the breast tissue. (Figs. 37 and 38.)

The drains are left open (Fig. 39) until the chest wall has been closed and the remaining upper lobe is reexpanded, at which time the drains will be clamped off.

Closure of the Chest. A length of doubled heavy silk is inserted as a pericostal suture below the seventh rib at about the midpoint of the wound. It is brought out above the fifth rib and the needle is cut out. This suture is anchored and not yet tied. A series of interrupted No. 2 nylon sutures are now placed in the margins of the periosteal bed and pleura. These sutures are individually anchored in clamps.

The edges of the wound are brought into apposition by pulling up and tying the heavy silk suture first inserted. The sutures inserted in the rib bed and pleura are tied individually; after this part of the closure has been completed, the heavy silk pericostal suture about the ribs is cut and removed. (Fig. 40.)

Interrupted nylon sutures are used to reconstruct the muscle wall. (Figs. 41 and 42.)

The skin incision is closed with interrupted cotton. (Fig. 43.)

The upper lobe is re-expanded by the

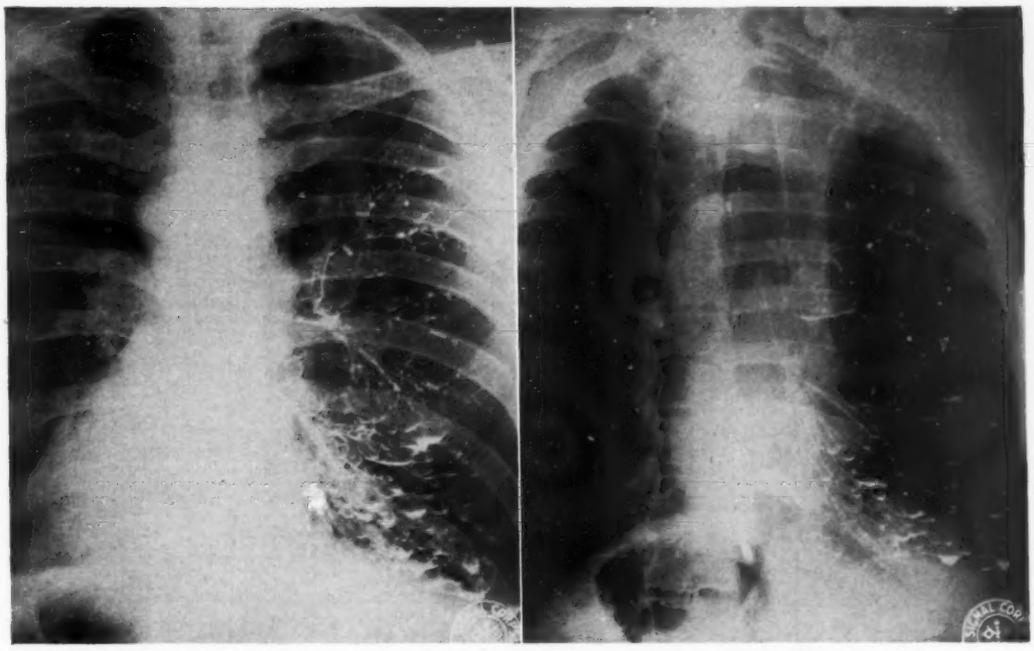

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Fig. 44. Postero-anterior x-ray study of chest following instillation of iodized oil into right bronchial tree.

Fig. 45. Oblique bronchogram of right chest.

Fig. 46. Right lateral bronchogram; involvement of the right middle and lower lobes is seen. In a subsequent preoperative study better filling of the upper lobe showed it to be uninvolved.

anesthetist, and the drains are clamped off until they are attached to tubes extending under water in gallon glass jars with two-hole stoppers.

If indications for bronchoscopy are present, this is done before the patient leaves the operating room.

CASE REPORT

The patient, a thirty-five year old Army officer, was admitted to the hospital February 6, 1946, as a transfer from another Army hospital with the diagnoses: (1) cyst, pulmonary, right lower lobe and (2) bronchiectasis, right lower lobe, severe.

The history revealed the usual diseases of childhood with complete recovery from each. In addition the patient had diphtheria at the age of three, pneumonia at the age of five and scarlet fever at the age of seven. The patient believed that the pneumonia was bilateral and he believed that some fluid was aspirated from the right chest. He made a complete and uncomplicated recovery from his illnesses otherwise, and there were no known residuals or sequelae. The officer went on active military duty on October 17, 1941.

About February 1, 1944, while on military

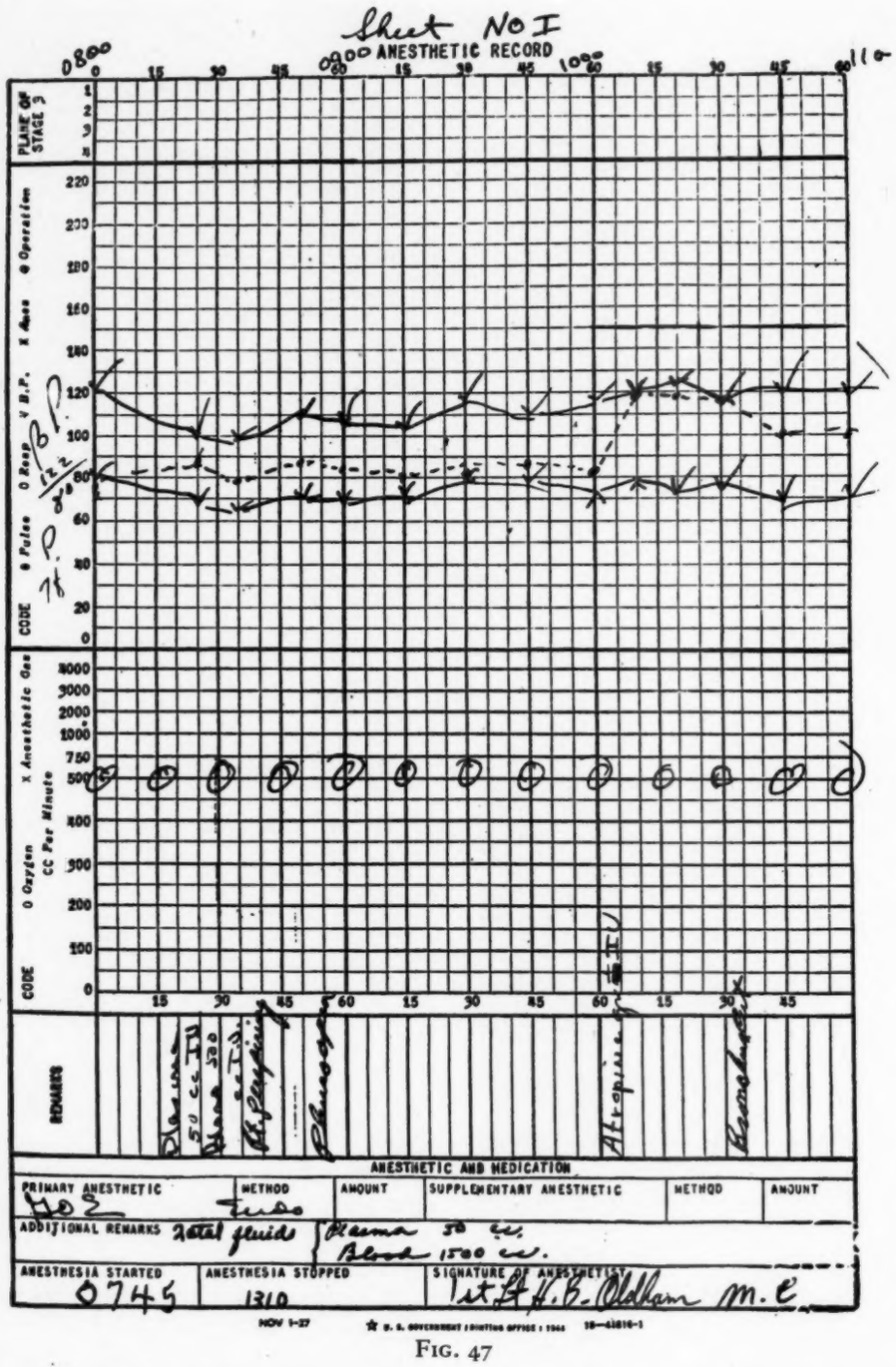


FIG. 47

FIGS. 47 and 48. Graphic record of anesthesia. The upper and lower solid lines, punctuated by V's, indicate the course of the patient's systolic and diastolic blood pressures. These started at 122/80 and were without significant change during the operation. The dotted line indicates the patient's pulse, starting at 80. The pulse was sharply accelerated to 120 at 10:00 o'clock (see top of graph) when

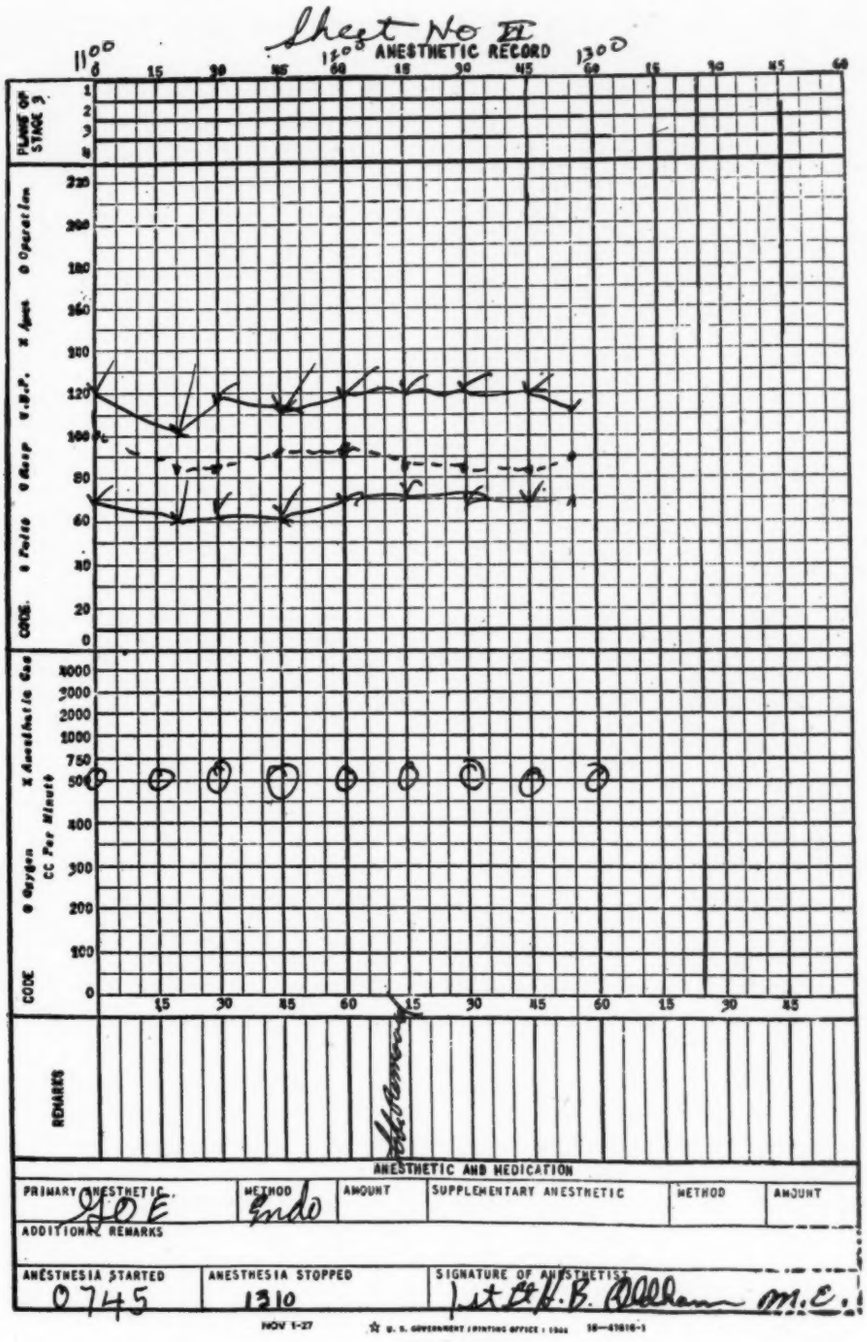


FIG. 48

atropine sulfate, 1/100 gr., was given intravenously as dissection of the bronchus was begun. The O's of the lower line indicate that the patient received 500 cc. of oxygen per minute during the operation, together with sufficient nitrous oxide and ether for adequate anesthesia.

Fig. 49. Dissection of specimen removed at surgery; numerous bronchiectatic, cyst-like lesions are seen. Microscopically, there is increase in connective tissue about the bronchioles with little evidence of acute inflammation.

duty in the Southwest Pacific, the patient noticed the onset of weight loss, shortness of breath, dizziness and feverishness. There was also a mild cough which was productive of a small amount of non-purulent sputum. With the persistence of these symptoms, the patient was first hospitalized about one week later. Treatment included the administration of oxygen, sulfonamides and bed rest. X-ray studies of the chest revealed evidences of bronchiectasis in the right lower lobe, but on May 25, 1944, he was sufficiently improved to return to duty. On June 30, 1944, the same symptoms which had previously occasioned hospitalization recurred except that at this time the cough was productive of 1 to 2 ounces of purulent sputum per day. The officer was again hospitalized on that date and after one week of further treatment with sulfonamides, he returned to duty improved. The cough and sputum persisted and climatic changes produced recurrences of fever and dizziness.

Blood began to appear in the sputum with a hemorrhage in August of 1945. Further hemorrhages occurred at intervals of four to six weeks, with continuous blood streaking between. The quantity of blood lost ranged up to 6 ounces and the quantity and foulness of the sputum were increased following these episodes. Bronchograms were made on August 20, 1945, and these were interpreted as showing bronchiectasis in the right middle and right lower lobes. The officer continued on duty until January 5, 1946, when, following his return to the United States, he entered a military hospital.

The patient's civilian occupation was athletic coach. The family history was non-contributory. There was a nasal operation in 1929 with no other surgery and no serious injuries.

Physical examination revealed a healthy appearing white male, 5 feet 11 inches tall, weighing 184 pounds. Physical examination of the chest revealed decreased tactile vocal fremitus in the right lower posterior chest, decreased breath sounds in the same area and dry râles throughout the right lower posterior chest with some inspiratory wheezes. The blood pressure was 128/98. In the remainder of the physical examination the findings were normal.

The blood Kahn was negative on February 7, 1946, and on the same date the white blood count was found to be 6,000 with 47 per cent neutrophiles, 50 per cent lymphocytes and 3 per cent eosinophiles. The hemoglobin was 104 per cent, 16.2 Gm. The urinalysis showed normal findings with the color yellow; specific gravity 1.026; character, clear; reaction, acid; albumin, negative; and sugar, negative. Examination of the sputum by smear and culture on February 11, 1946 was negative for acid-fast bacilli. The conventional x-ray study of the chest on February 7, 1946, was interpreted as revealing, "marked bullous emphysema in what appears to be the right middle lobe, with atelectasis and bronchiectasis involving the right lower lobe."

Bronchoscopy was performed on February 11, 1946. This revealed a generalized hyperemia of the tracheal and bronchial mucous membrane on both sides. The carina was somewhat thickened. There was a generalized distribution of a thin, mucoid type of secretion. There was no evidence of tumor, stenosis, granulation tissue or foreign body.

Bronchograms made on February 16, 1946, revealed the right middle and lower lobes to be honey-combed with bronchiectatic cysts. (Figs.

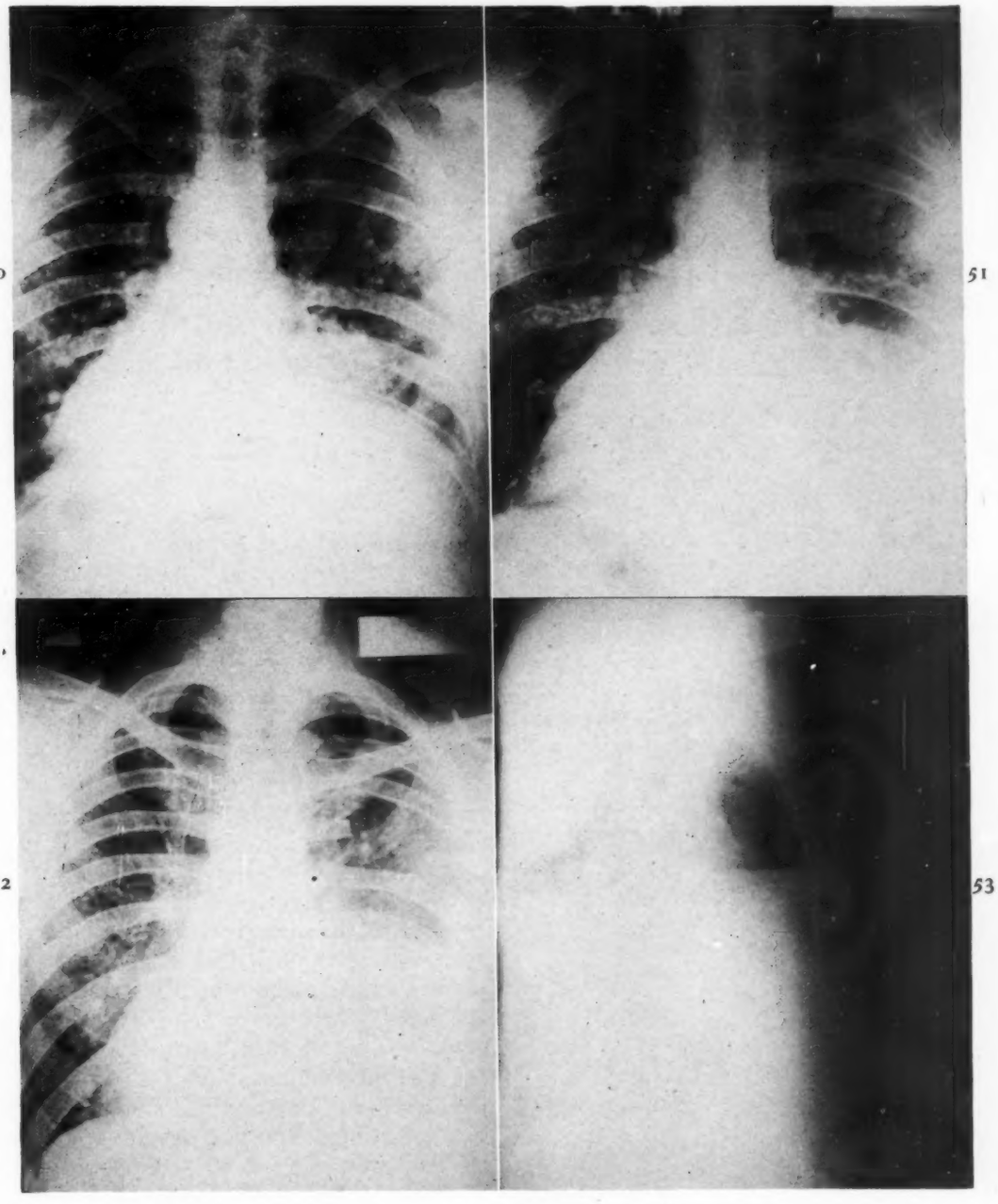
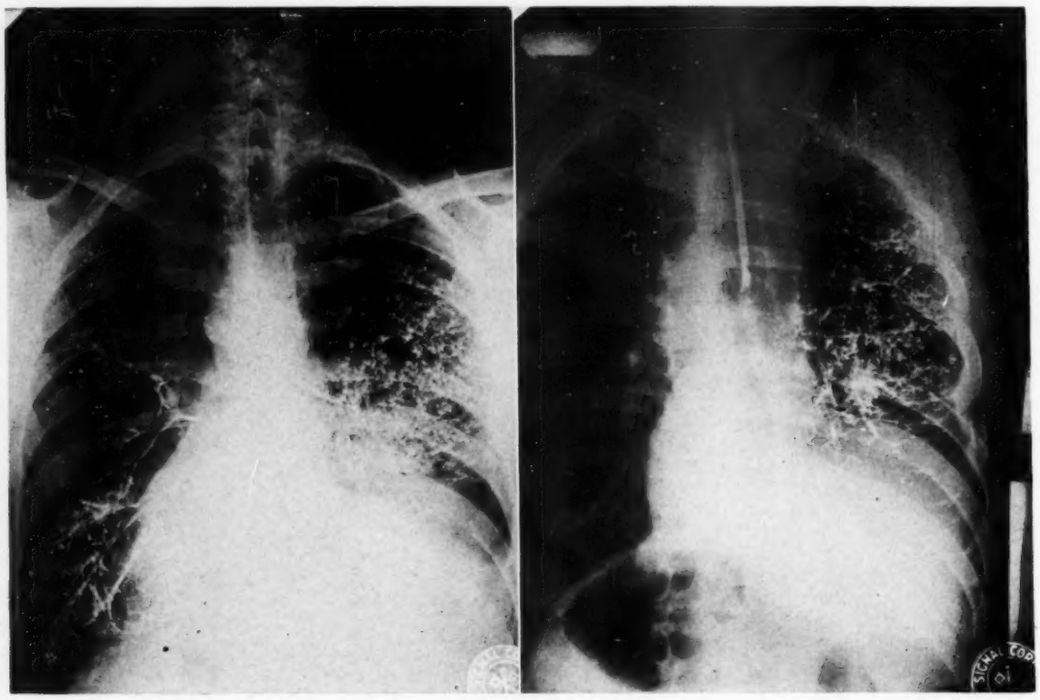


Fig. 50. Bedside postero-anterior x-ray of chest two days after lobectomy (April 5th). The rib resection and elevation of the right diaphragm are evident and a patch of possible bronchopneumonia appears at right base. Residual oil from the bronchographic studies remains.
Fig. 51. Bedside x-ray of chest on fourth postoperative day; pleural reaction appears quite marked; the pneumonic process is less evident.
Fig. 52. Fluid level right chest, seventh postoperative day (April 10th).
Fig. 53. Right lateral x-ray of chest showing fluid level (April 10th).

44, 45 and 46.) The remainder of the lung fields appeared to be normal. Repeat bronchograms were performed on February 27, 1946, to better demonstrate the right upper lobe and no involvement of this lobe was found. On February 18, 1946, the ears, nose and throat were again examined with normal findings, and on the same date a dental survey revealed the condition of the teeth and mouth to be satisfactory.

On March 25, 1946, following a leave, the patient was admitted to a surgical ward and was started on penicillin. This was given by



54 55

Fig. 54. Postoperative bronchogram, postero-anterior; the usual postlobectomy changes are present in the right chest with no evidence of residual bronchiectasis.
Fig. 55. Oblique bronchogram of right chest; the right upper lobe has expanded to fill the right chest.

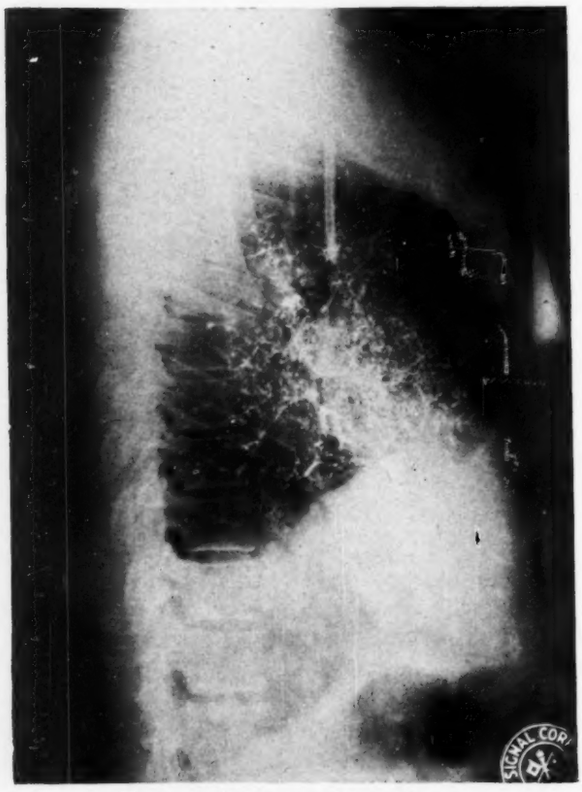


Fig. 56. Right lateral bronchogram.

intramuscular injection, 20,000 units every three hours.

On April 2, 1946, the patient was given 1½ gr. of nembutal at bed time. The blood which was Rh positive had been matched and typed, and blood suitable for transfusion was on hand. Three gr. of nembutal were given at 5:30 A.M. on April 3, 1946, followed by ¼ gr. of morphine sulfate and 1/100 gr. of atropine at 6:45 A.M. Operation was started at 8:20 A.M. under endotracheal gas, oxygen and ether.

The previously described operation for resection of the right middle and right lower lobes was carried out. (Figs. 47 and 48.) During the operation the blood pressure ranged within the following limits: systolic, 100 to 120; diastolic, 64 to 80. Plasma was started intravenously with the beginning of the operation. Fifteen minutes after the start of the operation the plasma intravenously was replaced by blood, and a total of 500 cc. of blood was given during the operation. Forty minutes after the start of the operation, the pleura was opened without noteworthy change in the blood pressure, pulse or respirations.

At 10:00 A.M. the patient was given 1/100 gr.

of atropine sulfate intravenously while the bronchus was being dissected. An acceleration of the pulse rate to 120 followed. At 10:30 the bronchus was cut. At 12:10 the lobes were removed from the chest (Fig. 49.), and at 12:55 the chest wound closure had been completed and the patient was bronchoscoped. There was a moderate amount of secretion in the tracheobronchial tree. After this had been removed by aspiration through the bronchoscope the patient was returned to the ward in good condition.

Immediately following the return of the patient to his room, he was placed in an oxygen tent. The drains from the chest which were connected with water-seal jars in surgery were checked at the bedside and were found in satisfactory working condition. Two cc. of demerol were given as needed for pain at intervals no closer than every three hours. The blood pressure and pulse rate were noted at half-hour intervals, and after the patient had aroused from the anesthesia at 7:30 P.M. he was sat up and "coughed" every two hours. The oxygen was discontinued at 10:30 P.M. The patient continued to receive penicillin on the same schedule as preoperatively.

As soon as the patient was able to swallow, he was started on sulfadiazine, 15 gr. (1 Gm). every four hours. This was continued for seven days. The patient's highest postoperative temperature was recorded on April 5th at which time the morning temperature was 99.6°F., pulse rate 92 and respirations 22. The following is the report of the radiologist on the bedside film on April 5, 1946 (Figure 50):

"Partial right rib resection. Probably incomplete expansion, remaining right lung which is difficult to evaluate on present bed-side study. Bronchopneumonic patch right cardiophrenic area. Soft tissue emphysema, right hemithorax laterally and in the cervical area. Residual opaque oil, left lung field. Moderate elevation, right diaphragm."

Bedside bronchoscopy was performed on April 6, 1946. Visualization of the right bronchial stump showed it to be healing satisfactorily, and a moderate quantity of mucosanguineous secretion was removed by aspiration. A bedside x-ray made the following day (Fig. 51) showed some improvement in the pneumonic process, but further x-ray studies made on April 10, 1946, (Figs. 52 and 53) showed the presence of pleural fluid on the right. The right chest was aspirated on April 11th, April 13th, April 15th and April 17th. On April 11th, 70 cc. of slightly blood-tinged serum was obtained. On April 13th, no fluid was obtained. On April 15th, 6 cc. was removed, and on April 15th the tap was again dry. Culture of the aspirated fluid revealed no growth. Bedside physical therapy was started on the first postoperative day and this was continued throughout the patient's convalescence.

On the patient's second postoperative day the drainage tubes were removed from the chest and the patient was allowed out of bed for short intervals. On the third postoperative day he was given bathroom privileges. The sutures were removed from the skin incision on the eighth postoperative day. On June 12, 1946, following the patient's return from a convalescent leave, bronchograms were again obtained and these revealed no evidence of residual bronchiectasis. (Figs. 54, 55 and 56.)

On August 1, 1946, the patient left the hospital for "temporary limited" duty with reexamination scheduled in about six months. At the time of discharge from the hospital the patient was free from cough and sputum, and he had no complaints other than mild shortness of breath and numbness of the skin anterior and inferior to the thoracotomy scar, both of which symptoms were gradually subsiding.

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FOUR HUNDRED CONSECUTIVE CASES OF JAUNDICE*

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TODAY there are two classifications of jaundice which have received widespread consideration, namely, those by Rich[1] and McNee.[2] Rich in his classification divides jaundice into the regurgitation and retention types while McNee, whose classification is the more widely accepted, uses three divisions, namely, obstructive, toxic and infectious and hemolytic.

The latter has been criticized by Rich who states that the classification of McNee is based upon both clinical and etiologic factors using two standards within one classification. His objection is rightly stated, but until Rich's classification receives more generalized usage the one of McNee's will remain.

In accord with Rich's classification is the one offered by Yater, in that he divides jaundice into the obstructive and hemolytic groups. He further subdivides the obstructive group into the intrahepatic and extrahepatic groups thereby placing toxic and infectious jaundice into the intrahepatic obstructive group in which they rightfully belong. This is also in accord with the teaching of Rich.

The accumulation of bile pigments within the circulation producing an icteric color of the sclera and skin may have various etiologic factors. Volumes have been written, numerous tests devised and still with the exception of a few basic accepted principles the entire question is far from being solved.

On the differential diagnosis of jaundice much also has been written and many tests have been devised, all of which have little significant value if the proper clinical observation has been made. There are still no tests which will replace the proper evaluation of a good history and physical examination. Certainly the daily observation of the stool as to color and the observation of urine will immediately give one a clue as to the type of jaundice with which the patient is afflicted. It is surely quite disconcerting to be called to see a patient with jaundice and see no daily observations recorded regarding the stool and urine.

The increase of the amount of bile in the urine or the changing color of the stool will afford pertinent information as to the advancement or retrogression of the etiologic factor producing jaundice. After the icteric changes of the skin have advanced to a certain point visual observation will give no further information as to the amount of bile pigment present in the circulation. And it is here that the icteric index or the serum bilirubinate determination is of some value. Nevertheless, even at this point, the observation of the stool and urine will still be of considerable diagnostic value. The assumption that the icteric index continues to rise to a given level as long as the stools are acholic is essentially correct. The changing color of the stool would definitely point to a fluctuating icteric index and would favor a diagnosis of lithogenic disease in place of pancreatic neoplasm.

The persistence of an acholic stool is significant of obstructive jaundice, be it intra- or extrahepatic in type. The release of the obstructive lesion would immediately manifest itself by a change in character of the urine and color of the stool, and would favor a diagnosis of lithogenic, toxic or infectious type of jaundice in preference to a neoplastic type.

The direct or indirect van den Bergh test has but little practical application if the

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aforementioned observations have been diligently carried out; for the acholic stool with choluria and a cholic stool with acholuria would immediately place the icterus in one of the two main divisions.

There are many other tests and while a few are of some value as confirmatory evidence, they all are usually found lacking when one seeks aid in the finer differential points. Since all the tests used for jaundice deal with liver function, the value is greatly depreciated by the fact that each test evaluates but one hepatic function and since the liver possesses many functions the impairment of any one function is not necessarily indicative of the function of the organ as a whole. Furthermore, the liver has marked regenerative powers and there must be considerable impairment of function present before it reflects itself in the laboratory analysis. One must also appreciate the fact that it is seldom he is dealing with one form of jaundice alone; for with the hemolytic type sooner or later hepatic impairment will become evident thereby complicating what once was a hemolytic jaundice with an intrahepatic obstructive or regurgitant type of jaundice. Also an intrahepatic or obstructive type of jaundice will soon produce liver changes thereby complicating the picture with a retention type of jaundice.

Jaundice has also been classified into the painless and painful type, the painful group suggesting an inflammatory or lithogenic basis while the painless type presumes a neoplastic or toxic basis. While the presence or absence of pain is of some value in differential diagnosis, nevertheless a study of patients suffering from icterus reveals a surprising number who die from painless obstruction of the common duct due to a stone or, conversely, those who because of pain were subjected to surgery in consequence of a diagnosis or a common duct stone only to find a malignant lesion in the pancreas.

It is not unusual to find at surgery or at autopsy a common duct stone as a cause of jaundice which had previously been diagnosed carcinoma of the head of the pancreas; conversely, a so-called common duct stone frequently proves to be a carcinoma of the head of the pancreas. Since this error is of frequent occurrence, it behooves one to perform an exploratory laparotomy in an effort to definitely determine the etiologic factor of the jaundice and to ameliorate or remove the fundamental cause of the affection if possible.

In this particular study we have reviewed the records of 400 consecutive patients suffering from jaundice as taken from the files of the Los Angeles County General Hospital. While a review of this type will produce statistics which are of some value in drawing conclusions, one must nevertheless be aware of the fact that due to the numerous uncontrollable variables encountered, namely, the failure to definitely establish all diagnosis through surgery or necropsy, we must be somewhat hesitant to accept the conclusions as being absolutely correct.

We must also recognize that diagnostic ability, even that of our best diagnostic institutions, is far from what might be desired. Furthermore, the large group of patients who have recovered and been discharged as having had a certain type of jaundice is no assurance that the diagnosis was correct. However, we are safe in assuming that patients so discharged as cured or improved were in their proper category as well as those who were dismissed in an unimproved condition and who showed a steady downhill course. But it must also be remembered that all patients with jaundice are not hospitalized.

In certain groups, namely, the lithogenic and neoplastic of the pancreas, sufficient patients were subjected to surgery or necropsy to correctly establish the diagnosis in a very high percentage of cases.

In presenting the following tables we have divided the four hundred patients into seven groups. (Table 1.) While we realize that the classification is far from ideal and certain rearrangement might be advisable, nevertheless, it at least presents

a working basis for presentation of the statistics.

In reviewing the group as a whole it immediately calls forth the observation that an individual who becomes jaundiced has approximately three and one-half of ten chances to recover. For as we appraise the mortality of the various groups it is quite apparent that the inflammatory group, which includes catarrhal jaundice, offers the patient the best opportunity to recover but in the remaining groups the mortality gradually increases, reaching a 100 per cent in the malignant group.

TABLE I

	Total	Per Cent
Malignancy	175*	43.75
Common duct stone	86	21.50
Inflammatory	64	16.00
Hemolytic	38	9.50
Portal cirrhosis	20	5.00
Unknown	11	2.75
Stricture	6	1.50
	400	100.00

* Includes two patients with Hodgkin's disease.

Malignancy. In this group we have one hundred seventy-five patients who suffered from malignancy, or 43.75 per cent of the total. This immediately brings the patient's chance of recovery to a 5.5 to 4.5 basis, or a little better than an equal break. (Table II.)

Carcinoma of the head of the pancreas leads the list as a cause of jaundice and accounts for approximately 25 per cent of cases, or about one in four. Coupled with the percentage of common duct stones, which is second in the list as a cause of jaundice with a total of 21.50 per cent, these two groups are responsible for approximately one-half of the jaundiced patients. (Table III.)

Neither medicine nor surgery has much to offer the malignant group. The presence of gallstones in a high percentage of malignant gallbladders offers a surgical incentive for reduction of the mortality for the removal of the gallbladder with its contained stones before the onset of malignancy would spare a high percentage of these patients. It is the only procedure which would give permanent relief for all other surgical procedures are only palliative in character.

TABLE II
MALIGNANCY

	Total	Per Cent of Malignancy	Per Cent of Total
Carcinoma of head of the pancreas	94	53.71	23.50
Carcinoma of biliary tract			
Gallbladder ducts and papilla of Vater	36	20.57	9.00
Metatastic Carcinoma to liver	32	18.29	8.00
Pressure on duct (two Hodgkin's disease)	12	6.86	3.00
Carcinoma of duodenum	1	.57	.25
	175	100.00	43.75

TABLE III

	No.	Per Cent of Total
Common duct stone	86	21.50

In twelve instances in the malignant group jaundice was ascribed to pressure on the biliary ducts by a malignant lesion including two cases of Hodgkin's disease. While this group represents the total in which extrahepatic ductal pressure produced jaundice, we must recognize the fact that the remaining number represent intrahepatic ductal pressure. It is generally conceded today that malignancy neither primary nor secondary will produce jaundice without pressure on the intra-or extrahepatic ducts. All of us no doubt have seen necropsies at which almost the entire liver was replaced by carcinomatous tissue and still no jaundice ensued. Carcinoma of the head of the pancreas if present can expect little but palliative care. While it is true that in recent years surgical

procedures have been devised for the removal of pancreatic malignancies, still the outcome at present, at least, is very disappointing. However, it is believed that with improvement of diagnostic means and surgical technic the outlook for these patients may become more hopeful.

Common Duct Stone. In the second group we are dealing with common duct stone. This group accounts for more than one-fifth of the entire series. From a medical and surgical standpoint it is groups two, three, four and six which offer the patient a little more than palliation, but in the inflammatory group, be it lithogenic or nonlithogenic, and the hemolytic group the patient can derive medical or surgical relief.

However, surgery for common duct stone in the presence of jaundice carries a high mortality. These patients usually are in the elderly age groups, frequently suffering from some concomitant disease, who have done nothing over the period of years to alleviate their suffering from gallbladder disease until jaundice appeared. Frequently these patients have been told, even when aware of the presence of stones, that surgical removal is not indicated unless they are subject to recurrent attacks of colic or inflammation.

There is no doubt that gallstones should be removed when first discovered. This applies equally to the gallbladder with the solitary or so-called silent stone as well as to the gallbladder filled with numerous pigmented or cholesterol stones with recurrent attacks of acute cholelithiasis and cholecystitis. While it is true that the solitary type of stone usually is evidence against a common duct stone, nevertheless this is a vicious type of stone which undoubtedly is responsible for many of the serious cases of jaundice due to ascending cholangitis and, incidentally, to many perforations.

Inflammatory Undoubtedly the most hopeful group in this series is the group suffering from catarrhal jaundice for here the recovery is 100 per cent. (Table IV.) Since there were no deaths and no surgeries, diagnosis rests solely on clinical observation and obviously upon the fact that the patient recovered. This disease is seldom found in the patient past thirty; it is usually in the twenty to thirty year group. One must be rather reluctant to make this diagnosis in an individual past fifty years of age. All patients in this group but two were below thirty-two years of age.

Table IV

	No.	Per Cent	Per Cent of Total
Catarrhal jaundice	31	48.44	7.75
Cholangitis	28		
non-calculous	16	25.00	4.00
calculous	12	18.75	3.00
Weil's disease	2	3.13	.50
Inflammatory edema	1	1.56	.25
Chronic pancreatitis	1	1.56	.25
Lues	1	1.56	.25

The diagnosis of this type of jaundice is usually readily established with a fair degree of certainty, without surgical or autopsy findings. The type of onset, age group, rapid course, mild gastrointestinal symptoms and the early recovery of the patient is the picture ascribed to catarrhal jaundice. Frequently time plays an essential part in establishing diagnosis.

Cholangitis, which is usually secondary to an inflammatory lesion of the extrahepatic biliary system, either calculous or non-calculous, comprised 43.75 per cent of this group or 7 per cent of the total. It is in this group, of course, that surgery is at its best, offering the patient a cure with but a low surgical mortality. Its presence can usually be diagnosed with a fair degree of accuracy because of the history of gallbladder disease or repeated attacks of biliary colic accompanied with a tender, upper right quadrant, temperature, chills and leukocytosis. If the patient is closely watched and surgery instituted at the proper time, low mortality will be encountered. The best prophylaxis, however, is removal of the gallbladder and its con-

tained stones at the proper time when the diagnosis is first established.

Two patients who suffered from Weil's disease and who recovered, one with inflammatory edema in which necropsy established the diagnosis, one suffering from chronic pancreatitis in which the diagnosis was established surgically and one patient with luetic jaundice comprise the remainder of this group.

TABLE V
TOXIC AND HEMOLYTIC ICTERUS

	No.	Per Cent	Per Cent of Total
Hemolytic	23	60.53	9.50
acquired	10	.....	2.50
congenital	13	.....	3.25
Toxic	12	31.58	3.00
arsphenamine	4		
phosphates	1		
sulfonamides	7		
Transfusion	2	5.26	.50
Massive pulmonary hemorrhage	1	2.63	.25

Toxic and Hemolytic Icterus. Toxic and hemolytic icterus composed thirty-eight of the total, or 9.50 per cent. (Table v.)

This probably represents the next most hopeful groups because a high percentage of these patients can be given medical or surgical relief or cure. It is well known that in congenital hemolytic icterus splenectomy will stem the progress of the disease. In the acquired type of hemolytic icterus the etiologic factor will determine the patient's possibility of receiving either medical or surgical aid. In approximately 50 per cent of these patients the diagnosis was confirmed by autopsy or surgery.

With the advent of sulphonamides, one must always be on the lookout for a toxic reaction for the possibility of the icterus being on a toxic basis does exist. But the ultimate outcome, if the drug is promptly discontinued and medical measures instituted, is quite satisfactory.

Arsphenamine reactions, as far as icterus is concerned, are seen less and less frequently because of improvement of technic and dosage. It is quite likely that with but few exceptions this type of jaundice will become less frequent. However, the question of permanent liver damage due to an overdose of an arsenical is always a possibility.

TABLE VI
PORTAL CIRRHOSIS

	No.	Per Cent of Total
Portal cirrhosis	20	500

In spite of the improvement of technic an occasional reaction from blood transfusions will occur. But with the advent of a wider knowledge of the Rh factor and proper grouping of the blood and cross matching, it is quite likely the icterus resulting from transfusion will rarely occur.

As a whole the outlook for a patient suffering from the toxic or hemolytic type of jaundice is quite encouraging.

Portal Cirrhosis. Portal cirrhosis represents 5 per cent of the total of the four hundred case histories reviewed. (Table VI.) Just why some patients suffering from this disease should become jaundiced and others should not remains a mystery. There is also some question as to the type of jaundice portal cirrhosis represents for the etiologic factor being unknown it is quite likely that it may be a mixed type of jaundice when present. The most accepted theory is that it is a nutritional deficiency secondary to alcohol or some unknown toxin. However, there are patients suffering from portal cirrhosis who have never used alcoholic beverages in any form; consequently, some other toxin beside alcohol must be considered as an etiologic factor. Suffice to say that the outlook for the group is poor and it is a question of only a few years when all will succumb to the disease.

Unknown. In the unknown group there were eleven, or 2.75 per cent of the total. (Table VII.) In reality this should be a

much larger group for frequently diagnosis was established for statistical purposes when a diagnosis of unknown etiology would have been more feasible. Naturally the patients that recover care little if the proper diagnosis has been established.

TABLE VII
UNKNOWN

	No.	Per Cent of Total
Unknown	11	2.75

On the other hand, autopsies in all deaths and surgical exploration if indicated, if the patient is willing, would probably bring many of these cases within their proper classification.

Strictures. The final group, namely, those of stricture again represent rather a hopeless picture. (Table VIII.) Naturally a congenital stricture of the newborn means a mortality of 100 per cent. As to the acquired type the surgeon will have to assume the entire responsibility for the mortality in this group. The presence of a stricture which is not promptly repaired will lead to cirrhotic changes in the liver which definitely contributes to the mortality. The patients are usually poor surgical risks; the secondary surgical procedures consume considerable time, leaving the patient in an extremely precarious condition postoperatively. This type of complication usually can be avoided. The proper exposure of an anatomic structure and careful ligating or severing of same will avoid most of these accidents. Since most accidents occur in removing the gallbladder in a retrograde manner, it behooves one to proceed with caution or still better remove the gallbladder from the fundus to the duct.

As long as the blood supply to the viscus has not been interrupted it may still be used for an anastomotic procedure, but with a preliminary ligation of the cystic artery the viscus must be removed for necrosis and gangrene are sure to follow.

TABLE VIII
STRICTURES

	No.	Per Cent of Total
Congenital	1	0.25
Acquired	5	1.25

SUMMARY

A patient who becomes jaundiced, regardless of age, has approximately three and one-half chances of ten to recover.

If the jaundice occurs in an individual who has reached middle age, his possible percentage of recovery is still smaller.

Removal of a gallbladder and its contained stones at the proper time after diagnosis has been established will reduce the number of deaths due to common duct stones, malignancy of the gallbladder and the inflammatory group due to calculous disease of the viscus.

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REVERSAL OF CIRCULATION OF THE BRAIN

PARTIAL REVERSAL IN FOUR HUMANS TREATED FOR PARALYSIS AND MENTAL CONDITIONS

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PARTIAL reversal of the circulation of the brain for therapeutic purposes can be performed with reasonable safety to the patient. All patients thus far operated upon were of the hopeless type with little chance for spontaneous recovery. Results given herein indicate that reestablishment of blood supply to parts of the brain by reversal or partial reversal of the circulation is a rational surgical procedure. While surgery of this general type has previously been performed experimentally on healthy animals and while many reversals of circulation have been performed on human limbs, it is believed that this paper gives the first reported work on reversal of circulation of the human brain or on reversal of brain circulation for improvement of mental or of paralytic conditions.

For proper understanding of the operation a discussion of the anatomic and physiologic relationships is desirable. The surgical technic developed by the writer will be found somewhat similar to previous methods but with a few significant innovations. Notable is the preliminary stretching of the artery. Without this it is difficult or impossible to perform the reversal, especially in patients of the elderly, sclerotic type for which this therapy may be indicated.

While this operation may be a drastic one for mere treatment of high blood pressure, it is noteworthy that in every case a definite lowering of the blood pressure resulted.

Blood Supply of the Brain. The arterial supply of the brain anastomoses considerably. The venous drainage is remarkable for its lack of symmetry. The capillaries of the brain possess considerable power of dilatation and contraction, more particularly under stimulation by carbon dioxide. To discuss brain disorders some detailed consideration of the brain circulation seems desirable.

Arteries of the Brain. The brain derives its blood supply symmetrically from the two internal carotid arteries and the two vertebral arteries. These four vessels unite in the anastomosis at the base of the brain known as the circle of Willis. From this circle arises part of the ganglionic system of brain arteries while the remainder of the ganglionic system and the cortical system of arteries arise as secondary branches that is, branches of arteries that are tributary from the circle.

Dorrance,[13] Saphir[24] and others have summarized literature on anomalies of the circle of Willis. In two cases of Saphir both posterior communicating arteries of the circle were either absent or small and without lumen. In his third case the right posterior communicating artery was absent, the right posterior cerebral artery receiving all its blood from the basilar artery and hence from the vertebral arteries. On the left side the left posterior cerebral artery was connected with the basilar artery only by a fibrous cord without recognizable lumen. It is, therefore, remarkable that this left posterior cerebral artery was supplied by an extra large, left posterior communicating artery. This, like many similar examples of arterial brain anomalies, indicates that the arteries of the brain have considerable adaptability when necessary.

While the circle of Willis undoubtedly aids the equalization of the arterial supply to the brain, there are many other important arterial anastomoses in the head. For example, Dorrance[13] claims that if a common carotid artery in man has been ligated, the flow through the corresponding internal carotid artery does not reverse but continues toward the brain although at about 50 per cent of the normal rate. The flow in the external carotid artery, however, has become retrograde as it must to be able to supply the internal carotid with blood. Thus, the external carotid artery can receive blood from the corresponding artery on the opposite side. Also, there are unilateral anastomoses which assist such as between the inferior and superior thyroid arteries.[2,13]

Poor development of one carotid artery should always be looked for before attempting surgery of these vessels. Homans[19] and Guinard[15] each had one of their patients die following ligation of a common carotid artery and autopsies showed the opposite carotid to be only weakly developed.

From the surgical standpoint it may be pointed out that the left common carotid artery arises directly from the aorta. On the right side it is the innominate artery that bifurcates to form the right subclavian artery and right common carotid artery. For these reasons the right common carotid artery is somewhat shorter and more difficult to manipulate than the left common carotid. Accordingly, the author's double arteriovenous anastomosis presents more technical difficulties when performed on the right side of the neck, and more stretching of the artery is necessary before severing. Hence, it is noteworthy that although based only on the four cases to be described presently there seems to be more improvement in the mental condition of the patient when the operation is performed on the right side rather than on the left.

Since the days of Cohnheim, anatomic books have taught that the circulation of the brain is peculiar in that the ganglionic system and cortical system of arteries do not anastomose and that the arterials of the two systems are "end arteries." Part of this error arose from the fact that it was based on study of the brains of non-human animals. Cobb[11,12] now states exactly the reverse for man: "There are no end arteries in the human brain. A red cell could travel from occipital to frontal pole if it had means of locomotion." Hence, a partial reversal of the brain circulation probably results, not in mere reversal of flow in circumscribed areas but rather, in a redistribution of blood flow throughout the entire intercommunicating network.

Even complete occlusion of both carotid arteries and both vertebral arteries need not result in death. As long ago as 1896, Hill[16,20] described such an "almost complete" occlusion in man resulting from a slow pathologic process without ill effects on the brain. More recently, Andreyev[2] found that in dogs, damaging effects on the brain were relatively slight if the four arteries were each cut and ligated several weeks apart. If all four were cut and ligated simultaneously, most animals died although a few might survive with considerable brain damage. The arterial anastomoses of the head and neck, therefore, possess considerable adaptability to take care of redistributed blood supply.

Veins of the Brain. The return flow of blood from the brain is not symmetrical. Many veins drain first into the venous sinuses which, because of their volume content, permit a limited expansion of brain tissue without increase of intracranial pressure. In most humans blood from the top of the brain flows to the superior sagittal sinus, eventually reaching the right jugular vein while blood from the deeper parts of the brain in which cerebral accidents are usually supposed to occur leaves through the left jugular vein. Therefore, unless the patient is believed to be one of those occasional subjects in whom the above arrangement is reversed, one may prefer first to reverse the circulation on the left side of the neck, particu-

larly since reversal may be performed on one side only if sufficient improvement results. Nevertheless, it will be further noted that of the four patients operated upon by the author by far the greatest improvement was found with the two who were operated upon on the right side although more cases are necessary to make this conclusive.

While the sinuses of the dura mater frequently communicate with each other, there is a distinct pathway of blood from each sinus destined to a certain internal jugular vein of the neck. The superior sagittal sinus commences at the foramen caecum, running backward near the top of the skull until it nears the internal occipital protuberance, deviates (usually) to the right and is continued as the right transverse sinus, finally becoming the right internal jugular vein. The inferior sagittal sinus courses backward in the central part of the skull, being named the straight sinus in its posterior part, and its blood then travels (usually) through the left transverse sinus to the left internal jugular vein.

The veins of the brain are devoid of valves. They leave the brain, pierce the arachnoid to empty into the various sinuses of the dura. The veins are named cerebral and cerebellar according to the region drained. The superior cerebral, middle cerebral and inferior cerebral veins drain external portions of the cerebrum; they empty into the superior sagittal, transverse, cavernous, sphenopartital and superior petrosal sinus. The great portion of the blood from this region of the brain passes thus (usually) into the right internal jugular vein.

The basal or deeper veins are formed by a union of the anterior cerebral, deep middle cerebral and inferior striate veins at the anterior perforated substance. These veins pass backward around the cerebral peduncles to empty into the internal cerebral veins or veins of Galen. These veins of Galen, which drain the deep parts of the hemispheres, are two in number. They are formed by union of the terminal vein with the choroid vein at the intraventricular foramen and course backward parallel with one another between the layers of the tela choroidea, receive the basal veins as previously stated, then unite to form the great vein of Galen and end in the straight sinus. Blood from this entire group and hence from the deeper parts of the cerebrum largely finds its way (usually) into the left internal jugular vein.

REVERSALS OF CIRCULATION

Previous Reversals of Circulation. Reversal of the circulation in limbs has been practiced for many years with some success. As long ago as 1912, Bernheim[6] collected records of fifty-two cases in the literature, including six of his own. Fifteen were "successful." Since many of these operations were alternatives to amputation, this may be a fair record.

Later, Bernheim[4,5] described a woman who suffered from gangrene of early Raynaud's disease and in whom he reversed the circulation in all four limbs. Eighteen years later the woman was still in fair condition.[5]

A partial reversal in the head of a dog was described by Horsley:[21] "The cardiac end of the common carotid was sutured to the cephalic end of the external jugular vein in a medium sized dog. This specimen was removed after 39 days. The sutures are distinctly buried, though the endothelium over them is still transparent at places."

Similarly, Guthrie[10] described a dog which lived for five years after the anastomoses between the cardiac end of the right common carotid artery and the cephalic end of the right external jugular vein, and also between the cephalic end of the right common carotid artery and the cardiac end of the right external jugular vein. The dog had excellent health and was eventually killed in a fight with other dogs. Guthrie stated after the dog's recovery, "That the pressure in the vein was less than arterial was indicated by the rela-

tively slight pressure on the cephalic portion of the vein adequate to occlude the lumen. . . . By palpation it could be demonstrated readily that the pressure in the cephalic end of the right external jugular vein was greater than in the unoperated vein."

Physiologic Arteriovenous Anastomoses. Partial reversal of the brain circulation must lead to some arterial blood reaching the vena cava without first traversing capillaries. It is, therefore, in point that arteriovenous anastomoses may exist normally in ears, fingers, toes, accessory sexual organs and perhaps elsewhere both in animals and in man. For details of anatomy and possible physiologic value of such anastomoses reviews such as that by Clark[10] are available. Whether or not arteriovenous anastomoses occur normally in the brain appears not certain as yet although there is some evidence for it.[3,33]

REVERSAL OF BRAIN CIRCULATION A NORMAL OCCURRENCE

While the brain requires a continuous, rich supply of oxygen, it probably never has the momentary enormous oxygen requirement of active muscle. Thus, the brain capillaries seem fairly long. Forbes[14] in early skull-window experiments "saw no capillaries," but did see long "arterioles" and "venules" which disappeared into the brain substance and in which blood corpuscles passed only "in single file." These vessels were no doubt actually long capillaries.

Forbes observed in some of these vessels that blood corpuscles might come to a stop and then, after some oscillation back and forth (synchronized with heart and respiratory pulsations), reverse their direction of flow. Therefore, reversal of circulation in the brain is to some extent a normal physiologic occurrence.

Pulsating Exophthalmos. A theoretically possible complication from reversal of the circulation of the brain is pulsating exophthalmos due to increased pressure in the cavernous sinus. This clinical entity has been described repeatedly, Martin and Mabon[28] claiming to have collected references to 812 cases from the literature. In all or nearly all these cases the internal carotid artery had burst or broken into the cavernous sinus. The internal carotid artery is essentially surrounded by venous blood in that sinus. Thus, exuded blood from the artery rapidly flows away through venous channels and does not have an opportunity to clot and check the hemorrhage. Exophthalmos is caused by the blood pressure developed in the cavernous sinus.

In the author's patients no such pulsation or exophthalmos developed. This was true even in his single case (Case II) in which the reversal was performed on both sides of the neck. While exophthalmos is always a possibility, it is likely that reversal of the circulation is less serious from this standpoint than mere bursting of the internal carotid artery into the cavernous sinus. The latter condition short-circuits the blood thus giving rise to possible anemia or ischemia in addition to the increased pressure. Generally it is found that when venous channels carry arterial blood under pressure and if there is no disturbance of nutrition, the vessel walls tend to thicken sufficiently to hold the arterial pressure. Thus, it may well be that for the same increase of pressure in the cavernous sinus, the danger of exophthalmos is less with a reversal of circulation than with the mere short-circuiting of blood from a ruptured internal carotid artery. The appearance and subsequent clearing of pain in the right eye following operation on the patient in Case I tends to support this hypothesis.

Clinic Pathologic Phase. Ischemia is defined by a medical dictionary as "a local anemia due to mechanical obstruction usually from contraction of a blood vessel," but for the purpose of this article ischemia will be defined as the condition in a circumscribed area in which there is a diminished nutritive activity resulting in total or partial dysfunction of the cells involved. The disorder, therefore, may

vary from simple diminished function to complete necrosis of the cells.

The etiologic factors resulting in ischemic conditions in the brain are many, but the pathologic condition of the cells involved is essentially the same. While the onset of ischemia may be sudden and dramatic as in a "stroke," Alvarez[1] recently suggested that senile degeneration may result from gradual formation of tiny clots in the arterioles of the brain.

Blood-gas determinations indicate that the oxygen content of venous blood leaving the brain is more nearly constant than in venous blood returning from muscular tissue. Lennox[24] quotes data, including some of his own, which indicate that in many instances the oxygen content of venous blood leaving muscular tissue may be greater than of blood in the jugular vein. Exactly the opposite is claimed by Haldane[17] who states that muscular tissue consumes up to 80 per cent of the oxygen circulating through it while brain tissue consumes perhaps one-half as much of the circulating oxygen. The cases of Lennox were mostly humans subject to seizures, but determinations were made after "lying down fifteen minutes or longer," hence the muscle tissue of the patients in Lennox's cases required very little oxygen. In any event, results of both Lennox and Haldane are compatible with the hypothesis that while oxygen content of blood leaving muscle tissue may vary within wide limits, oxygen content in the cerebral veins is normally maintained at a fairly constant level.

Hence, any disturbance, circulatory or otherwise, which slows the flow of blood in all or in part of the brain may have serious effects. The cortical cells particularly undergo necrosis within a few minutes after complete or nearly complete anemia is established. Recovery may be possible if the anemia is not too nearly complete or not of too long duration.

The problem, then, is alleviation of ischemia with its panphasic pathologic consequences. A solution, therefore, is the rehemotization of the area or areas involved.

Improvement of Epileptics from Circulation Reversal. Epileptic or other convulsions are often regarded as pathologic but in a sense convulsions are quasi normal. Asphyxia, strychnine or other drugs may produce convulsions in normal individuals. Consider the sleeping rabbit whose fur is slightly ruffled by the breath of a fox. The rabbit may instantly spring high into the air and then run away. The initial spring differs only in degree and duration from a pathologic convulsion yet it saves the animal's life. Similarly, if a person is suddenly burned at some point, there is an immediate involuntary movement to escape; such a movement is not greatly different from a Jacksonian fit, except that it serves an obviously useful purpose. Vomiting and sneezing may similarly be regarded as (at least often) useful convulsions.

In general terms, therefore, a convulsion is a sudden mobilization of the nerve cells to meet an emergency but it is directed by the subconscious mind. (Nevertheless, Freud and others have regarded tantrums, hysteria, etc., as sometimes conscious or at least semiconscious, especially if a useful purpose is served.) "If, however, we were to hazard a two-word guess concerning the fundamental element in the precipitation of seizures in epilepsy, it would be oxygen-lack."[25] Whether or not the seizure may partially relieve such oxygen lack, need not be discussed at this point.

If epileptic convulsions in a particular patient arise from or at least are precipitated by oxygen-lack, improvement is evidently possible as a result of the author's operation for reversal of brain circulation. Results to date with his single epileptic patient (Case IV) tend to support this prediction.

VARIABILITY OF BRAIN CIRCULATION

From Mental Work. Various parts of the brain appear to enjoy a substantial increase in circulation as well as increase in

number of injectable capillaries when that part of the brain is working. This has been found in the olfactory lobe of the brain after smelling ammonia, also in the visual tracts of the brain after a light has been flashed into the eye.[36] Again, performance of "mental arithmetic" increases cerebral blood flow.[26] This general principle is the basis of the "lie detector" which actually measures systemic blood pressure and is used in police investigations.

From Nervous Control. Therefore, the cerebral arteries and even capillaries according to recent investigations have the power to dilate or contract for control of blood flow. Nerve fibers have been traced along the arterial walls. It has been stated that these fibers are found on arteries 50 microns or more in diameter[3] or even 25 microns or more in diameter.[11] "In the pia, even the capillaries appear to be innervated."[11]

Thus, the brain vessels do not just passively carry whatever amount of blood the heart pumps to them. Wolff[35] states that when one common carotid artery in the dog is tied off, blood flow in the opposite internal carotid artery increases nearly 100 per cent. Increase in intracranial pressure would be expected to force blood out of the cranial vessels thus constricting them. While this indeed happens with very high intracranial pressures approaching the arterial pressure, more moderate pressures cause the cerebral arteries to dilate even if the systemic blood pressure has not increased.[36] Again, local mechanical pressure on a small portion of the brain may cause dilatation of vessels in that portion without affecting vessels in the remainder of the brain as shown by skull-window experiments of Wolff and collaborators.[36]

Forbes[3,36] made an often quoted statement regarding arteries of diameter of 50 microns or more when their sympathetic nerves are electrically stimulated at some distance from the artery itself. His statement is to the effect that cerebral arteries contract about 8 per cent of their diameter while systemic arteries (as in the skin) contract about 80 per cent of their diameters. Some have concluded from this that cerebral arteries have little power of contraction, but if the electrical stimulation be applied directly to the wall of the cerebral artery itself practically 100 per cent contraction or almost complete occlusion results.[36]

Again, although flow of blood in the pial arteries slows when the common carotid arteries are both simultaneously occluded, there is an immediate increase in diameter although from the fall of pressure in these pial arteries a contraction would be expected on mechanical grounds alone. If within one-half a minute the carotids are released, the pial arteries dilate still more, even sufficiently to cause diapedesis.[36] Therefore, it appears that when it is necessary to maintain nutrition of the brain, the cerebral arteries can and do enlarge considerably.

From Carbon Dioxide. Under usual conditions the nutritional requirements of the brain do not change within such wide limits as is the case between active and resting muscle or between perspiring and cold skin. It is probably for this reason that many physiologic experiments, such as those just considered, have led some to conclude that cerebral blood vessels are relatively passive and do not dilate or contract excessively.

Lowered oxygen content, or especially increased carbon dioxide content of the blood, obviously must greatly increase the requirements of the brain for blood since the brain prefers to work under fairly uniform concentrations of those two chemical substances. Carbon dioxide appears, from various nerve section and other experiments, to have a powerful, direct action on the vessel walls themselves, even on the brain capillaries.[3,36] Carbon dioxide, therefore, seems to have a strong stimulating effect in every part of the brain, not merely in the so-called "respiratory center." Since the effect remains after extensive nerve section and thus appears to be at

least partly a direct effect, it has been referred to as "humoral" action.

For example, decreased ventilation of the lungs causes simultaneous increase in carbon dioxide content and decrease in oxygen content of the blood. Cerebral vasodilatation under these conditions may be so extreme as to increase the cerebral blood flow as much as four times, but with no change in flow through the external carotid artery, and actual decrease in blood flow through the leg.[36] This dilatation of the cerebral vessels is easily seen through skull-windows. Ether also appears to have a strong dilating effect on cerebral vessels[3,36] which agrees with the practical fact that ether has held well against all competitors as a safe anesthetic. So-called improvements are often only combinations of basal or induction or other anesthetics with ether.

It is apparent that if the brain circulation is really reversed completely the arteries would contain venous blood with its high carbon dioxide content and low oxygen content. Thus, the humoral reflexes would then tend to increase greatly the caliber of the arteries. This might not only permit release of clots or other obstructions, but in any event should permit increased blood flow since the veins, owing to their relatively thin walls, should not impede the circulation to the extent that is possible in sclerotic arteries. Veins do thicken eventually when carrying arterial blood under pressure, but should carry the blood for some years before becoming sclerotic. Hence, it appears that reversal of the brain circulation provides a possible means for substantial increase of the blood flow through the brain, and if high blood pressure in a patient is caused by resistance of sclerotic cerebral arteries, reversal of brain circulation may well permit reduction of the arterial pressure.

RELATION OF PARTIAL REVERSAL OF CIRCULATION TO THE CAROTID SINUS

In recent years, importance has been given the carotid sinus reflexes by occidental medicine. Strangely enough, oriental ju-jitsu experts have known of these reflexes for many centuries.[23] They know that either a sharp blow or severe pressure against the carotid sinus (not just against the side of the neck or against the vagus nerve) may produce unconsciousness within a second or so. Men are uncomfortable when wearing high, stiff collars and some may faint when wearing them; this is probably due to irritation of the carotid sinus.[3]

Similarly, Dorrance[13] states that ligation of the internal carotid artery is more dangerous than ligation of the common carotid and explains this as partly a carotid-sinus effect. Ligation of the internal carotid artery tends to increase pressure within the sinus which is located at the point where the common carotid artery bifurcates to form the external and internal carotid arteries. Thus, reflexes are started which still further reduce the brain circulation, but ligation of the common carotid artery obviously reduces the intrasinal blood pressure, thus tending to dilate the cerebral arteries. In the author's operation the pressure in the carotid sinus on the operated sinus is necessarily reduced thus also tending to dilate cerebral arteries.

It is true that elaborate physiologic experiments by Heymans[18] and others, using attempts to control arterial pressure by head perfusion at constant pressure, have not uniformly shown a direct effect of pressure changes in the carotid sinus on cerebral artery caliber.[3] In any event, in the intact animal, pressure decrease in the carotid sinus tends both to raise the systemic blood pressure and also to constrict the peripheral vessels, hence the final result is to increase considerably the cerebral blood flow.[3] Skull-window experiments have shown that a fall in the systemic blood pressure produces dilatation of the cerebral vessels although, of course, fall of pressure in the carotid sinus does not institute all the reflexes concerned.[3]

The carotid sinus is also sensitive to increased carbon dioxide content of the

blood.[3] In the author's double arteriovenous anastomosis, venous blood (containing more carbon dioxide than arterial blood) is introduced into the carotid sinus on the operated side. Hence, reflexes are started which tend to improve the volume of the cerebral circulation.

PRINCIPLES OF BLOOD VESSEL SURGERY

Blood vessel surgery differs fundamentally from most other kinds of surgery. The severed vessels, instead of being ligated as is usual, must be opened and sutured. Thus, there is great chance for hemorrhage. Because of the damage to the blood vessels, together with the shock of operation, clotting within the operated vessels is always a danger. For that reason many surgeons recommend anticoagulants such as heparin in blood vessel surgery even though this increases the danger of later or secondary hemorrhage.

In the larger vessels, such as the common carotid artery and the internal jugular vein, the chance of clotting within the vessels is relatively reduced because of the rapid blood flow. Furthermore, the internal surface area of a vessel is proportional to its diameter, but the internal volume is proportional to the square of the diameter. Hence, in larger vessels there is proportionately less damaged endothelium to cause clotting of the blood but danger from hemorrhage is greater in larger vessels; therefore, also based on the writer's practical experience, he does not use heparin or other anticoagulants in surgery of the larger vessels.

There are two basic methods of uniting ends of blood vessels: end-to-end suture methods first popularized by Carrel[7,9,27] and invagination methods in which one vessel is slipped inside the second vessel. End-to-end methods may have the disadvantage that thread remains in contact with the flowing blood although Carrel reduced this effect by careful coating of the silk threads with petroleum oil to reduce clotting. Also, there are numerous holes made by the needle which open directly into the blood stream and these offer chance for secondary hemorrhage, especially if infection develops. It is true that by the method of Zaaijer[32,37] the sutures may be placed so that when tightened the ends of the vessels automatically dilate as flanges; no thread remains in the flowing blood but the vessels must then be flexible, as for example, veins. Furthermore, end-to-end methods lend themselves most readily to union of two vessels of about the same size, but the common carotid artery in man is much smaller than the internal jugular vein; it seems thus more logical to slip one vessel inside the other.

In the invagination method, the principle used by the writer, one vessel stump having perhaps been turned back over itself is slipped into the second vessel stump. This has the advantage that no needle holes need open into the blood stream directly, also that flowing blood need not necessarily touch any blood vessel substance except endothelium thus reducing danger of clotting inside the vessels. The turning back process has been omitted partly because it may damage the vessel. Furthermore, in most of the author's patients with a certain amount of arteriosclerosis, turning back of the sclerotic carotid artery is almost impossible, if not entirely so. The first successful union of two arteries in a human, by Murphy,[29] was by an invagination method.

Later workers with this method have introduced various sorts of rings made of ivory,[30] of pigeon bone,[35] of magnesium,[21,31] of Vitallium[8] or of silver or other substances to assist in the turning back process. Since such a ring is usually left in the wound as part of the anastomosis, its material may be chosen either for inertness, as with Vitallium, or for absorbability as with magnesium.

Removable rings of various types have also been used which are generally supplied with handles; these may be removed as soon as suturing of the vessels is completed.

FIG. 1. Diagram of right side of neck before operation showing carotid artery (to right) and internal jugular vein (to left).

Among such are the Crile cannula and the Elsberg monkey wrench cannula.[7] These were popular when temporary arteriovenous anastomoses were used between two persons for blood transfusions and are less suitable for permanent anastomoses.

The internal jugular vein when severed and devoid of blood is difficult to control because of the thinness of its walls. The writer has not yet used a Vitallium tube, a Crile cannula or a similar device with human patients but believes that such might facilitate handling of the vein. After the operation is completed, however, the tube or cannula would not be left as part of the anastomosis. It would necessarily be removed since it would have been used to support the larger vessel rather than the smaller one.

Operative Procedure. The patient is prepared in the normal manner and under local anesthesia. An incision is made along the anterior border of the sterno-cleido-mastoideus muscle through the integuments, superficial and deep fascia. The internal jugular vein and common carotid artery are dissected free of fascia, care being taken to avoid undue irritation of the vagus nerve or of the carotid sinus. The vagus nerve is carefully pushed aside. (Fig. 1.)

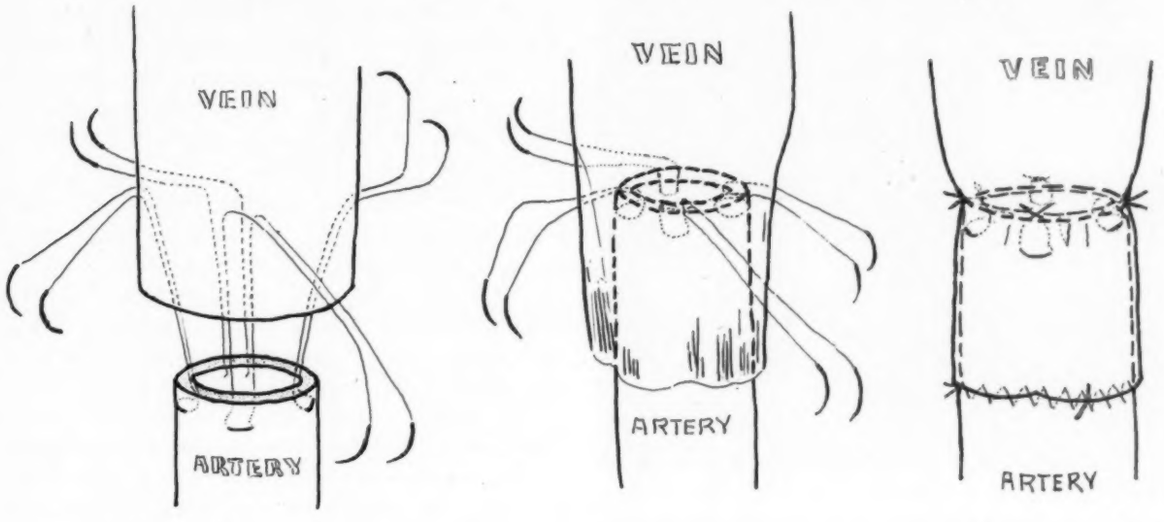
All communicating branches of the internal jugular vein are doubly ligated close to the vein and severed. There are generally two of these but sometimes one or three. There is a large communicating vein between the external jugular and internal jugular veins. The author has doubly ligated this vein as part of the operation, except that in one or two cases with blood pressures around 240 mm. it seemed best to leave this as an anastomosis to relieve the excessive blood pressure. The common carotid artery has no branches until it bifurcates into the internal and external carotid arteries at the carotid sinus. This usually takes place at the upper border of the thyroid cartilage.

At this stage of the operation the common carotid artery is now stretched very gently until it is fairly loose and has a tendency to be longer than necessary. This is a very important step without which serious accident may occur. Previous workers have often begun to make a double arteriovenous anastomosis and had to be content with a single anastomosis, perhaps with transplantation of a portion of vein to fill a gap; proper stretching of the artery will avoid such embarrassments.

The clamps are then placed on the common carotid artery and on the internal jugular vein and the vessels are severed. An end-to-end anastomosis is effected first between the cardiac part of the carotid artery and the cephalic part of the internal jugular vein. Then a separate anastomosis is effected between the cardiac part of the internal jugular vein and the cephalic part of the common carotid artery. To avoid tedious repetition in description of the cases to follow and in other parts of the paper this operation is referred to simply as a double arteriovenous anastomosis, that is, it is so defined for the purposes of this paper. After the anastomoses are effected the clamps are released slowly and on the vein first; all

leaks are repaired before full arterial pressure is admitted.

The writer's technic for the anastomosis has been worked out by him on dogs. The animals recover well after the double anastomosis or double reversal of circulation has been performed on both sides of the neck.



2 3 4

Fig. 2. General arrangement of four preliminary sutures. This much is somewhat similar to the arrangement used by Murphy[29] except that he used three sutures (six needles) and also started from *inside* the artery instead of from outside. Also he joined two arterial stumps. It may be desirable to use more spacing between the ends of a suture, in the vein, than shown above.

Fig. 3. Four preliminary sutures after drawing or inserting artery into vein. Since the vein is quite flexible, there is very little suture material in contact with flowing blood in the final anastomosis.

Fig. 4. Final anastomosis. Preliminary sutures have been tied, additional sutures placed on the sleeve of the vein to tack it to the artery and further sutures added wherever needed to repair leaks or to strengthen the anastomosis.

The internal jugular veins of the human being are much larger than the common carotid arteries. In the dog the difference is not so great. The technic used can be easily understood if reference is made to the illustrations. (Figs. 2 to 4.) After the artery is severed a mattress suture is placed in the artery from without inward, using a double-needle suture. The needles are brought out through the opening or lumen of the artery as close as possible to the cut edge of the arterial endothelium. This is to give as much strength as possible to the suture without undue exposure of suture material to blood flow in the finished anastomosis.

Four such sutures (with eight needles) are placed in the artery symmetrically. The needles are then forced through the vein from within outward in the proper symmetrical positions and the artery is inserted into the vein. The sutures are simultaneously pulled tightly and then tied. The posterior one is tied first, otherwise it would be hard to reach.

Next, a continuous suture is placed on the sleeve or cut end of the vein, including the arterial wall except for its endothelium. This gives a sort of double set of sutures. This seems important in the junction of such large vessels. Upon removal of the clamps any leakage is controlled by further suture and pressure. The wound is then closed by suturing all anatomic layers as is usual in neck surgery. (Fig. 5.)

An immediately apparent effect after the operation is that the patient becomes cyanotic to a greater or lesser degree for a few hours to a few days. Then the cyanosis gradually clears. The cyanosis, however, occurs only in the face and not the body, being due to disturbance and interruption of the normal flow of blood to and from the head. Hence, the normal oxygen and

FIG. 5. Diagram of right side of neck showing carotid artery and internal jugular vein after double arteriovenous anastomosis.

carbon dioxide balances of the blood to the brain are disturbed. Since increased blood carbon dioxide causes dilatation to vessels of the brain and since greater flow of blood through the brain is necessary for the relief of ischemic conditions, these cyanotic effects are perhaps desirable.

The patient complains of some headache and pain following operation but the inconvenience is surprisingly little. There is some apprehension and the patient complains of some pounding in the ears. A definite bruit is audible for some weeks over the internal jugular vein at the point where the arterial blood enters it. Some patients can hear this sound for at least some weeks after the operation, even after the pounding in the ears has ceased.

The objective symptoms include cyanosis at first, followed by clearing of this condition, and also a marked drop in systolic blood pressure. It may be noted that the operated patients were usually hypertensive and there was a definite lowering of the systolic blood pressure which remained lowered.

CASE REPORTS

CASE 1. I. D., aged fifty-six, was admitted to St. Agnes Hospital on April 20, 1944, with the following history: She stated that following a dizzy spell on November 11, 1943, she suddenly became unable to lift her left arm. The following day she became completely paralyzed on the left side of her body. Her physician stated that although she was treated both at home and in a hospital, there had been no noticeable improvement.

The author found a heavy set, middle-aged woman with a blood pressure of 210/110 and complete paralysis of the left side, with inability to move either her left arm or her left foot. There was chronic left sinusitis with thickened membrane and a trace of albumin in the urine as well as some pus.

On April 21, 1944, under local anesthesia, a right double arteriovenous anastomosis was made between the common carotid artery and the internal jugular vein of the right side. The patient returned to her room in good condition, although with slight cyanosis of the face, and was kept well narcotized for twenty-four hours. On April 23rd the blood pressure was down to 150/95, the cyanosis appeared cleared and the patient felt fairly well although she had some pain in the left side of her head. On April 25th the patient complained quite severely of pain in the right eye, although a fundus examination revealed no disorder. By April 28th the pain in both the right eye and the left side of her head had disappeared. The transient pain in the right eye may have been related to "pulsating exophthalmos," a possibility previously discussed.

On May 2nd she appeared bright and cheerful and all stitches were removed. On May 4th she moved her left leg for the first time in six months and was able to do so at will. On May 8th she was dismissed from the hospital; two weeks later she was able to get out of bed under her own power. In 1946, she was able to get around her house and do light housework. Her left leg and arm were spastic but usable; she was able to get around with the aid of one crutch. Her blood pressure still remained around 150 systolic. At the present time this patient is feeling very well, has gained considerable weight and is able to get around the house. She still wears a brace on her left ankle for support and uses a crutch whenever she has to be on her feet for any length of time. Blood

pressure is now 160/100. There has been no further improvement of the arm and very little if any of the leg since the last report.

Case II. O. D., aged sixty-one, was admitted to St. Agnes Hospital on July 25, 1945, with the following history: She had been given shock treatment by another physician for correction of a mental disease. Following treatment, she became completely paralyzed on the right side and lost her speech. Her physical condition was average; blood pressure was 165/80. She refused to eat and had to be fed by a stomach tube.

On August 4th under local anesthesia, a left double arteriovenous anastomosis was made between the common carotid artery and the internal jugular vein of the left side. The patient returned to her room in good condition, except for some cyanosis of the face. She was kept well narcotized for twenty-four hours, after which the cyanosis cleared. On August 6th her blood pressure was down to 130/90. On August 16th the sutures were removed but the mental condition seemed unimproved, and she could not speak. By August 25th the patient responded to questions better by shaking or nodding her head. Also on this date she was, for the first time since the shock treatment, able to take food by herself instead of with aid of a stomach tube. Improvement continued to September 4th, at which time her blood pressure was 150/90.

On September 5th operation was performed. The patient stood the operation well although cyanosis again developed; she returned to her room in good condition. Three hours later obstructive breathing occurred. The patient expired, autopsy was refused and the cause of death was not known. From the nurse's report it appeared that there may have been acute edema of the larynx.

Case III. W. B., aged sixty-four, was admitted to St. Agnes Hospital November 30, 1944, with the following history as told by relatives: On September 25, 1944, he became suddenly ill and lost his speech. Later that evening he became paralyzed on the right side. Although treated both at home and in the hospital, there had been no noticeable improvement. The author found a man of small stature who appeared to be older than the age stated. He presented tortuous blood vessels in the temple and in the bend of the elbow. All the vessels appeared sclerotic; the blood pressure was 188/100. The right half of the body appeared to be completely paralyzed.

On December 2, 1944, under local anesthesia, a left double arteriovenous anastomosis was made between the common carotid artery and the internal jugular vein of the left side. The artery was extremely sclerotic; one-half of the lumen was occluded to give a typical pipestem vessel. After operation the patient appeared to be in good condition except for cyanosis of the face which continued for about three days. The patient returned home on January 2, 1945. His speech and ability to move his paralyzed limbs had not improved. However, he seemed to respond better to questions than before operation. While before operation he had to be fed by mouth, he now took nourishment by himself and was apparently feeling much better.

Both before and after operation the patient suffered from severe uremic conditions. This patient expired five months after operation. The cause of death was unknown, as the patient had left the vicinity.

Case IV. R. C., aged fifteen, was admitted to St. Agnes Hospital on October 30, 1946, with the following history according to his mother: His early childhood was not remarkable; he had chicken pox and measles. Epileptic seizures began at five years of age of a very light, static staring type which lasted generally only a few seconds. They continued to be mild, although with some increasing severity, until he fell 18 feet from a tree in June, 1943. He was unconscious twenty-four hours and hospitalized five days. The seizures then became more severe, often with unconsciousness for many minutes, that is, he would not remember afterward what happened during the seizures. If walking when a seizure appeared, he might fall to the ground with a typical grand mal.

Beginning in June, 1943 and until October 31, 1946, he might have from five to seven epileptic seizures daily, with typical tonic and clonic convulsions lasting from a few seconds to twenty minutes. About three times weekly he had a typical grand mal seizure.

In addition, during the three-year period, he had eight or ten spells in which he would go into a semicomatose condition lasting from one-half hour to three days, the longest spell being about two and one-half years ago. During these spells he could walk around but did not

know where he was or what he was doing. There were no convulsions during these spells but each was always followed after a day or so by a very severe epileptic seizure.

Beginning at five years of age, treatment was given with ½ gr. doses of phenobarbital three times daily, but seizures still gradually increased in intensity and frequency although they would be less frequent during actual phenobarbital dosage. Following the fall, phenobarbital in 1½ gr. doses three times daily, later three daily doses each of 1½ gr. dilantin daily and still later three daily doses each of 5 gr. tridione, seemed of little help. Treatment in various clinics and hospitals was tried without apparent result.

From October 15, 1946 to November 1, 1946, he took each day: 1½ gr. phenobarbital, three 1½ gr. dilantin capsules and three 5 gr. tridione capsules daily. In spite of this he had a very severe epileptic seizure on October 30, 1946.

When first seen by the author on October 31, 1946, thorough physical examination revealed little abnormal, except that all reflexes were apparently exaggerated. Blood pressure was 140/85 and the red count was 4,750,000 per cu. mm.

On November 1, 1946, a right double arteriovenous anastomosis of the common carotid artery and internal jugular vein of the right side was performed. Immediately following operation there was slight cyanosis of the face which lasted only a few hours. The patient stood the operation well and was kept well narcotized for four days. Barbiturates were then gradually reduced and were stopped altogether on November 11, 1946. Two days later the patient returned home.

Shortly thereafter he had a rather severe epileptic convulsion although not as severe as previous to operation. He is now taking three 1½ gr. capsules of dilantin daily. Immediately following the operation, blood pressure dropped to 120 systolic and has remained there.

Daily reports from his mother indicate that, so far, there appears to be marked improvement. Instead of several seizures daily there are now about three weekly. Also, the boy states that since the operation, he does not become completely unconscious during a seizure, that is, afterward he is able to recall what others were doing during the seizure. During the week December 1 to 7, 1946, two of the three seizures were mild and hardly noticeable. The third, while more severe, did not cause unconsciousness.

About December 1, 1946, the boy was able to return to school; he had been unable to attend school except for one month during the past years (1946) because of the epileptic attacks. This young man at the time of operation was fifteen years of age, his weight was 147 pounds and height was 5 feet 11½ inches. At the present time he is sixteen years of age, weighs 175 pounds and is 6 feet 3 inches tall. He has been attending school regularly since December, 1946, takes part in physical education activities and has discontinued taking any kind of medicine.

Seizures, which occur occasionally, are much lighter; he does not fall or become unconscious. Intervals between seizures have been as long as thirty days.

About May, 1947, there was a noticeable dilatation of the anterior facial vein. This dilatation became progressively worse and more noticeable until July, 1947, at which time he was reoperated upon with ligation of the common facial vein. The patient left the hospital on the fourth day. This condition was completely cured.

COMMENTS

Cases II and III showed the least improvement, a fact that was first ascribed to the patients' greater age and generally poorer condition at time of operation. In particular, the patient described in Case III had arteries so hardened and thickened that operation was rather difficult. Nevertheless, it is true that these patients were operated upon on the left side while the best cases, I and IV, were operated upon on the right side. Thus, it may well be that the right side should be the choice for operation.

As already remarked, operation on this side presents greater technical difficulty because of the shortness and other anatomic features of the right common carotid artery. Discussion of the anatomic phase showed that arterial blood introduced into the right internal jugular vein should find its way into the superior sagittal sinus. Thus, the veins supplied with

arterial blood should be the ones that are observed in skull-window experiments rather than the deeper veins. The highly successful double anastomosis of Guthrie in a dog[16] was on the right side.

Furthermore, the report of Andreyev[2] on dogs with the two carotid and two vertebral arteries ligated indicates that after all it may be the surface region of the brain hemispheres that is first to lose its blood supply. Thus, in an injection autopsy of such a dog:[2] "The arteries on the convex surface of the hemispheres did not contain any of the opaque substance." This would be the general region that could be supplied with arterial blood through the right jugular vein. Because of the limited number of cases presented here, however, further speculation on choice of right or left side for the operation is deferred.

The fall in blood pressure after operation, which was from 210 to 150 systolic in Case 1, is immediate and dramatic. While high blood pressure in some patients is believed to arise from kidney dysfunction, it may also be postulated that if there is a region of the brain with circulatory deficiency, the blood pressure may rise in an effort to restore the blood flow in that region. Support for this hypothesis is found in a statement by Alvarez:[1] "When with a suspicious episode a high blood pressure drops to normal I look on this happening as strongly supportive of the view that there was a small stroke, because I have seen this happen so many times." In his case VII, for example,[1] following one of her "spells," blood pressure dropped immediately from 200 to 135 systolic and remained there. In this patient it is likely that there had been circulatory difficulty in a region of the brain but that a sudden cerebral accident destroyed the circulation so completely that further attempts by the heart to maintain circulation there were useless, hence the blood pressure dropped to normal. It is postulated that in the author's cases when nutrition of ischemic areas was restored by circulation reversal, the need for high blood pressure may similarly disappear, although by restoration rather than by destruction of the ischemic region. But with either the type of "small stroke" described by Alvarez or with circulation reversal, there being sometimes no further need for the high blood pressure, it can fall to normal.

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DEVELOPMENT OF ULTRAVIOLET BLOOD IRRADIATION

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THE development of ultraviolet blood irradiation therapy was an outgrowth of an attempt to utilize the bactericidal properties of ultraviolet rays in the treatment of blood stream infections. If the greatest advantage was to be derived from the bactericidal properties of the rays, a method of applying them directly to the blood stream of the patient had to be developed. The problem faced was twofold in nature: (1) to develop a method of application and (2) to determine the necessary dosage.

The first step taken was to review the literature on ultraviolet irradiation in general to determine what, if anything, had been accomplished along this line and the extent of success or failure. The literature, mostly from European sources, was voluminous and seemed to establish the fact that some researchers in the field believed that most of the systemic reactions observed following exposure of the skin to ultraviolet rays were due to the influence of the rays upon the blood.

It was noted in the literature that the source of the ultraviolet energy was inconstant and varied widely. This fact alone would produce variable results and often a failure to obtain any measurable results. A source of ultraviolet of known intensity was sought that could be easily controlled so that a uniform dosage could be achieved and duplicated at will. The Burdick water-cooled ultraviolet generator was selected as having the desired characteristics.

It was believed that a method of exposing the blood directly was feasible. An irradiation chamber was developed by Knott and Edblom to allow direct exposure of the blood. The irradiation chamber was circular and approximately 2 inches in diameter and about 1 inch thick. The inside of the chamber had a labyrinthian passage connecting the inlet and outlet formed by baffle plates that were ground to fit flush against the quartz window that formed the top of the chamber. Thus the blood must flow through the labyrinth by passing around one end of each baffle plate instead of across them. The labyrinth was approximately 1 cm. deep.

The irradiation chamber was so designed as to provide maximum turbulence of the blood to accomplish two desired effects: (1) to prevent the formation of a film of blood forming on the chamber window that would absorb and filter out much of the desired wave lengths of ultraviolet. In an opaque substance, such as blood, it is necessary to turbulate the substance to prevent the formation of a film of blood that absorbs the ultraviolet and prevents it from reaching the flowing substance. (2) To insure that all parts of the blood be equally exposed to prevent overexposure of some parts and underexposure of others. If the blood flowing through the chamber is not turbulated, a portion of it remains next to the window and absorbs the ultraviolet thus preventing uniform exposure of the total volume and resulting in the overexposure of one portion and underexposure of others. The development of the irradiation chamber provided a method of exposing blood uniformly so that all parts were equally exposed and that a uniform and repeatable dosage could be administered.

The first step taken in the original work by Knott and Edblom was to determine roughly the tolerance of the red blood cells to ultraviolet rays when directly exposed in whole blood. Small quantities of whole blood were placed in a quartz test tube and citrated 1 to 5 with a 2½ per cent solution of sodium citrate. With

the ultraviolet generator operating at 60 volts and 4+ amperes supplying wave lengths 2399 to 3650–54 Angstrom units, the blood samples were exposed at contact with the source of ultraviolet from 0 to 280 seconds in five-second and ten-second graduations of exposure. After each interval the specimens were microscopically examined to note any change in the form of the red blood cells. No change was noted up to seventy seconds and then a slight poikilocytosis occurred. This condition disappeared after eighty seconds and no further change was noted up to 280 seconds when disintegration took place.

This same procedure was followed with whole citrated blood inoculated with cultures of Staphlyococcus aureus. Cultures were taken after each interval. Growth of the organisms occurred only in the first three samples with no growth showing in any sample exposed more than ten seconds. These experiments were repeated until it was demonstrated that the bactericidal properties of the rays were effective well within the tolerance range of the red cells.

The next step was to undertake animal experiments. Strains of Staphlyococcus aureus and hemolytic streptococcus of known virulence to dogs were used. These organisms were injected in gross amounts intravenously into dogs and cultures taken periodically to insure that acute septicemia developed. The animals were then irradiated by tapping two veins and pumping the blood from one to the other through the irradiation chamber. The pumping was accomplished with a Luer syringe and a three-way valve. The process was continued until the estimated total volume of the animal's blood had been irradiated. As the blood passed through the irradiation chamber, it was subjected to ultraviolet irradiation from a Burdick water-cooled ultraviolet generator held in contact with the chamber, the actual source of the ultraviolet being approximately 3 cm. from the chamber window. The test animals all died on the fifth to seventh day from what appeared to be a combination of profound depression, a progressive respiratory slow-up and failure. All the animals at the time of death had negative blood cultures, thus indicating that the organisms had been destroyed, a marked contrast to the control animals in whom death was preceded by an overwhelming septicemia. The literature suggested that overexposure to ultraviolet rays could conceivably produce a profound depressive effect.

It was then attempted to determine the susceptibility or tolerance of the host to ultraviolet rays impinged directly upon the blood stream. This was approached by two methods: (1) To irradiate the entire blood stream, but with reduced dosages to determine the maximum the animal could stand, and (2) to maintain the dosage of the earlier experiment and to reduce the total volume irradiated.

The latter method had been suggested by an incident in which the apparatus had failed and only partial irradiation had been accomplished; the test animal in this instance survived and negative blood cultures were achieved. Both methods were pursued until it was established that the second method was subject to less mechanical difficulty and productive of more uniform results.

From these experiments it was found that it was unnecessary to expose the blood sufficiently to kill the bacteria directly. It was also noted that it evidently was unnecessary to expose all of the blood to achieve a desired effect upon the entire blood stream. The total amount of blood to be irradiated was determined to be $\frac{1}{16}$ to $\frac{1}{20}$ of the estimated blood volume or approximately $1\frac{1}{2}$ cc. per pound of body weight. Continued work, using the same virulent bacteria, established the fact that ten test animals treated by the newer system, all recovered from an overwhelming infection and that none showed any ill effects. All dogs were carefully checked with blood counts and continued observation; no untoward results were noted after four months in most of the animals, and

after four years in the case of one to which we became attached sentimentally. Four untreated dogs died of septicemia.

From observations made on the work done on animals a corrected or extended hypothesis was established. The evidence accumulated so far seemed to indicate that the impingement of small, carefully regulated doses of selected ultraviolet rays directly upon the blood stream of test animals in some way increased the bactericidal properties of the blood stream; that by this method of treatment some influence was exerted upon the vital resistance factors of the host, enabling it to overcome the infection and to progress to complete recovery; that some influence was exerted upon the toxic conditions produced by the disease, either by direct action upon the toxins themselves or by indirect action on the toxins through the host's rallied defense mechanisms. To what extent any of the above is true or false was not determined from the data available from the work done to this point. The literature searched earlier indicated that certain known toxins had been inactivated directly by ultraviolet rays under given conditions, but whether and to what extent it was possible to vary the conditions and still produce the result was not known, nor was it known whether such a detoxification effect could be produced *in vivo* as well as *in vitro*. The work on animals was carried on until it was definitely determined that the animals suffered no ill effects from the treatment and yet showed a clinical detoxification *in vivo*.

The first treatment on a human took place in 1928. Consultation held the patient to be in a moribund state from septic abortion complicated by hemolytic streptococcus septicemia. Ultraviolet blood irradiation therapy was instituted as a last resort to see if the septicemia in a human could be affected. The patient responded to treatment and proceeded to an uneventful recovery. She has since borne two children, and when last checked in 1940 was found to be in good health with a normal urinalysis and blood count. Nothing further was done in the treatment of human patients until 1933.

In the meanwhile some work was done in Chicago at a private research foundation where experiments were conducted to try to determine what particular component of the blood was most profoundly affected by ultraviolet irradiation and in what manner. This work had to be curtailed without definite results being obtained because of the difficulty of following the chain chemical reactions set up and the lack of equipment and money to pursue them to conclusion. To the author's knowledge, technics have not as yet been devised to determine the end result or the modus operandi of chain chemical reactions set up by the stimuli of ultraviolet rays to such a complex compound as human blood. No conclusions were drawn as to the results obtained in Chicago. Clinical observations seemed to us to be the most important procedure to follow next until sufficient knowledge could be accumulated from the use of the procedure upon which to base problems of basic research.

This first became possible in 1933. Virgil K. Hancock, M.D., of Seattle, Washington, with James Tate Mason, M.D., a consultant, felt justified in using the procedure as a last resort on a patient, apparently moribund, with advanced hemolytic streptococcus septicemia. This case was described in detail by Hancock and Knott[1] in the first published article reporting this technic. Successful conclusion of the case led to further use in similar cases of serious nature in which it was believed the patient was in a moribund state.

In this work with Hancock, daily blood counts and cultures were taken on all patients treated. Careful study of these blood counts revealed that in cases of overwhelming infection, when leukopenia was present, there was following ultraviolet blood irradiation a rise in the white cell count to a degree consistent with the severity of the infection, the count then

diminishing with clinical improvement. An increase was also noted in the red cell count in patients suffering from hemolytic streptococcus septicemia. Consistent responses were achieved in the treatment of septicemia, but it was noted that streptococcus blood stream infections were more easily controlled than were staphylococcus, the latter requiring usually more than one treatment, while the former usually responded favorably to one. In a large majority of the cases, a marked cyanosis was present at the time of initiation of ultraviolet blood irradiation. It was noted that during the treatment or immediately following the treatment a very rapid relief of the cyanosis occurred with a corresponding easement of respiratory embarrassment. This response was consistent and was, in a majority of cases, accompanied by a noticeable flushing or reddening of the skin with a distinct loss of pallor. It was not determined at first whether this phenomenon was due to a greater assimilation of oxygen or a vasodilation effect, or both.

The easing of respiratory embarrassment that occurred consistently in septicemia led to the application of the procedure in cases of well defined and well diagnosed pneumonia. In a series of seventy-five cases in which the diagnoses were confirmed by x-rays, all patients were treated by this method and, according to Hancock, the method was found to be extremely valuable in combatting the pneumonia. The course of the disease following ultraviolet blood irradiation was marked by a rapid fall in temperature, disappearance of cyanosis, often within three to five minutes, cessation of delirium if present, a rapid subsidence of toxic symptoms generally, a marked reduction in pulse rate and a rapid resolution of consolidation. A shortening of hospitalization and convalescence occurred regularly. This procedure was later found by Miley to be especially valuable in the treatment of virus type pneumonia in which chemotherapy had proven of doubtful value.

The consistent results obtained in treatment of cases of septicemia gave rise to the question as to the modus operandi of irradiated blood. In an endeavor to find out the process of sterilization of the blood stream, opsonic index studies were made to see if the phagocytic power of the polymorphonuclears was influenced by ultraviolet blood irradiation. These studies were started in 1936. When proper exposure *in vitro* had been determined, it was noted that the average increase in the bacterial ingestion by polymorphonuclears was 50 per cent plus. This work was repeated in 1946 with improved technic and the ingestion of bacteria by polymorphonuclears was found to be increased up to 78 per cent. It was also determined that there was very little latitude between the proper exposure at which the polymorphonuclears were stimulated to a maximum degree of ingestion and the amount of exposure at which they were destroyed. Destruction of the polymorphonuclears was characterized by a blowing out of the surface of the cells. Although bacteria were present within the cell, the cell was destroyed on one surface. It was not determined just how this destruction took place but it is significant that when the proper exposure was exceeded by 25 to 40 per cent such destruction consistently took place. The general literature on the subject cites examples of this phenomenon on cells from overexposure.

From the information up to this point and considering the clinical experiments, personal experience and observation, the technic was improved, and this improved technic is known as the "Knott Technic of Ultraviolet Blood Irradiation." The irradiation chamber was also redesigned to give a more thoroughly uniform exposure to the blood and became the Knott Hemo-Irradiation Chamber now in use.

A number of irradiation units were made up and placed in the hands of physicians interested in the procedure so that wide clinical data could be accumulated. It is noteworthy that none of the physicians

using the procedure in different parts of the country found any cause from their observations to disagree with the early hypothesis that had been set up to govern its use. It is also significant that all the physicians using the procedure enjoyed parallel results in like conditions as long as the prescribed technic was strictly adhered to. It was determined from the work done that the established technic was not subject to variation. While the technic was simple, it had to be adhered to strictly. Any variation at all would produce variable results. Investigations that led to the development of the established technic revealed that exposure factors have to be varied as the media exposed are changed, i.e., whole blood requires different handling than does plasma. The depth of the channel in the exposure chamber is a critical factor. Exposures of culture media for the production of vaccines necessitates a different chamber construction and a corresponding change in the time factor to achieve the desired results. The established Knott technic of ultraviolet blood irradiation therapy was worked out for the exposure of whole blood and was arrived at by correlating all of the factors of energy, time cell tolerance and turbulation of the blood. In all media being irradiated it was noted, as set forth in the literature, that intermittent exposure required less total exposure time than a continuous exposure to produce the same end result. Intermittent exposure also facilitates a more uniform dosage being administered.

Dr. George P. Miley, of New York, was one of the early workers with the procedure after Knott improved the chamber and released them to physicians for further accumulation of clinical data. Miley became interested in the oxygen exchange factor, suspected and commented upon by Knott. He was particularly impressed by the rapid disappearance of cyanosis following treatment. Miley made extensive studies on the combined oxygen values of venous blood before and after irradiation in pathologic states. This work[2] was published in June, 1939, and showed a general increase in the combined oxygen value of venous blood following irradiation.

Another early worker, Dr. E. W. Rebbeck, and his co-worker, Glassburn, of the Shadyside Hospital in Pittsburgh, became interested in the relief of toxic symptoms following treatment and surmised that if such was the case, it should be reflected in the lipoidal fraction of the blood and show a change in the chylomicron picture. They made interesting observations in the chylomicron study of about 100 patients. They observed that in advanced pathologic states the clumping of the blood fats was restored to a normal state, both *in vivo* and *in vitro* immediately following the exposure of the blood to the Knott technic, i.e., they noted the dispersal and breaking up of the blood fat clumps present in a toxic patients' blood, plus a return to normal of the lost Brownian movement of lipoid particles, the fats indicating a return to normal of the whole chylomicron picture. As the Brownian movement of the blood fats return, there is often but not always a return to normal of the blood sedimentation rate and a decrease of the toxic symptoms. This latter result is most consistent and is noticeable within twenty-four to forty-eight hours following treatment. Usually a marked drop of temperature to normal is evidenced concomitantly.

The procedure has been used clinically and reported upon by several workers following Hancock and Knott's[1] article on the treatment of infections with the method. Barrett[3,4] discusses five years' experience and reported on irradiation of blood with ultraviolet spectral energy in two articles. Miley[2,5-14] published articles on the use of the procedure in the treatment of thrombophlebitis,[5] staphylococcemias,[6,9] botulism,[7] poliomyelitis,[8] non-healing wound,[10] acute pyogenic infection,[11,14] and Miley and co-workers have reported its use in peritonitis,[15] acute infections[16] and asthma.[17,18]

In 1947 Miley reported the use of this method in seventy-nine consecutive cases of acute virus and virus-like infections, stating that he believed ultraviolet blood irradiation therapy could be relied upon consistently to control an infection of a virus or virus-like nature in a safe and efficient manner. In discussing this paper Barger gave a report of twenty-three cases of bulbar poliomyelitis and six cases of spinal poliomyelitis in which he definitely believed he could confirm Miley's observations. In addition Barger quoted in detail three cases of advanced encephalitis in which he had used ultraviolet blood irradiation, stating that in each case following this type of treatment a rapid disappearance of all signs and symptoms of encephalitis occurred, and that no residuals were apparent after from six months to three years.

Rebbeck has reported its use in Escherichia coli septicemia,[21] postabortional sepsis,[22] puerperal sepsis,[23] and its use preoperatively,[20] and Rebbeck and co-workers reported on peritonitis[15] and double septicemia.[19] Rebbeck and Lewis[30] have found in nine consecutive cases of typhoid fever that in six patients who received ultraviolet blood irradiation there was a remarkably rapid recovery as contrasted with three control cases in which one death occurred.

Olney has reported on the treatment of pelvic cellulitis[24] and biliary disease[25] with ultraviolet blood irradiation. Hancock[26] discusses its use in blood stream infections. Davidson[27] further discusses oxygen reaction in anoxia and bends. Sullivan and Beroza[28] discuss short time irradiation on biochemical compounds and the influence on phagocytosis.

From the published reports of the clinical use of the procedure evidence has been accumulated upon which to found further basic research and investigation.

Up to 1940 the administration of ultraviolet blood irradiation had been carried on by a gravity and manual method of flow and timing. Such a method of application, of course, left a margin for human error, as the operator had to observe the rate of flow and time exposure as well as other factors in the irradiation treatment. To eliminate this margin of error, an apparatus was designed to control mechanically all the factors of irradiation and insure dosage that could be repeated at will. This apparatus now known as the Knott Hemo-Irradiator was first displayed at the Scientific Exhibit Section of the American Medical Association's meeting in New York in 1940.

The apparatus consists of a self-contained, water-cooled, ultraviolet generator and an electrically driven transfusion pump and exposure device that mechanically controls the rate of flow and exposure of the blood to the rays. The apparatus can be accurately controlled as to the amount of ultraviolet generated as well as the time of exposure of the blood to the ultraviolet ray. Between the source of the ultraviolet rays and the blood irradiation chamber there is a revolving shutter that subjects the blood to intermittent exposure to the rays. The turbulence within the chamber augmented by the pump causes the blood to turbulate against the quartz window of the chamber so that a uniform exposure of all parts of the blood is insured.

Since development of the Knott Hemo-Irradiator, clinical application of the procedure has greatly increased. Since 1923, when ultraviolet irradiation of the blood was first conceived, approximately 45,000 treatments have been administered in various pathologic conditions. The field of use has widened materially from the original application of the procedure in the treatment of blood stream infections, as experience and research indicated that it would be adaptable to pathologic conditions other than blood stream infections. As a result of extensive clinical experience and a certain amount of basic research, workers in this field are generally agreed that the favorable clinical results observed are due to many biochemical and physio-

logic effects: notably an increase in blood oxygen absorption, a rise in phagocytic action and general immunologic status, the setting up of an efficient detoxifying mechanism, and increased rate and volume of blood flow through the capillary circulation and beneficial alteration chemically of certain important constituents of the blood, e.g., adrenalin, dihydroxy-phenylalanine, cysteine hydrochloride, glutathione and ergosterol.

Thus ultraviolet blood irradiation therapy must be considered as a powerful therapeutic agent which the skilled physician and surgeon can use in a wide variety of disease processes, since in using this method one is applying certain fundamental biochemical and physiologic effects.

The favorable results experienced from the clinical use of the ultraviolet blood irradiation certainly warrants extensive investigation of the modus operandi of the procedure and detailed, intensive clinical studies to determine fully its potentialities.

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GASTRIC DIVERTICULA*

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A REVIEW of the available medical literature impresses one with the apparent infrequency of gastric diverticula, in fact, most articles on this subject refer in their opening paragraphs to the rarity of such an abnormality and the lack of clinical and pathologic experience with this specific condition. However, with the rapid improvement in roentgenologic technic, an ever increasing number of reported cases are accumulating, most of which have appeared in publications during the past twenty-five years. The paucity of reports prior to this period can be explained on the basis of the fact that it is almost impossible to make a preoperative diagnosis of a gastric diverticulum without the aid of x-ray study.

It appears to be the consensus of medical opinion that a fairly high percentage of such diverticula are asymptomatic and are discovered quite by accident during routine surgical, postmortem or roentgen-ray examination. Furthermore, the surgeon who has had operative experience with this condition will concede that many gastric diverticula are overlooked in the operating room as well as in the postmortem room. An out-pouching of the stomach that appears quite obvious when filled with barium has the facility of remarkable contraction when empty. One may safely assume that all proven cases of diverticula of the stomach have not been reported, perhaps among other reasons because they are considered by some to be of little clinical significance.

Granted that this abnormality is uncommon it still appears that it is not as rare as the literature would lead us to believe. Unfortunately, insufficient data has been accumulated to permit a thorough evaluation of the diverticulum in reference to gastric symptomatology and therapy. In the interests of further clarifying the issue this paper is presented. Two new case reports are included with surgical verification of their preoperative diagnoses.

Fournier[1] in 1774 is credited with first describing gastric diverticula as a pathologic entity and Helmont,[2,3] in 1804 is said to have reported the first case of such an abnormality. Martin[4] in his extensive study of the historical background of this subject refutes these statements as false and adds the correction that Thomas Baillie in 1793 reported the earliest authentic case. Brown[5] first demonstrated by x-ray a gastric diverticulum preoperatively in 1916; however, he misdiagnosed the findings to be those of an ulcer. Akerlund[6] in 1923 gave great impetus to the interest in this condition when he published a monograph on the roentgenologic aspect of diverticula of the stomach.

By 1923 Tupper[7] found thirty-eight cases reported in the literature and a little more than a decade later Rivers, Stevens and Kirklin[3] presented fourteen proven cases which they added to the 141 cases mentioned in their report. One year later Martin[4] wrote that "an exhaustive search through the Archives of the History Department of the Johns Hopkins Hospital was unfruitful in revealing a single case diagnosed ante mortem. Not one was reported after laparotomy. Apparently none was reported by the roentgenologic department. The search was continued through the records of the pathology department and five cases were found."

Tracey[8] in 1943 reviewed the cases from

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the files of the Lahey Clinic where on the basis of roentgenologic findings, the diagnosis of diverticula of the stomach had been made in thirty-five patients. More recent reports have been presented by Hunter,[9] Frank,[10] Paulson[11] and Telford.[12] Bockus[13] in his book on gastroenterology estimated that more than 150 cases of gastric diverticula had been reported prior to 1943.

Classification. The greater portion of discussion found in literature with reference to the classification of gastric diverticula is of theoretical importance only and much of this is confusing to say the least. One of the more standard[3] classifications subdivides these abnormalities as follows: (1) True diverticula—those in which all layers of the gastric wall are present without definite evidence that organic disease played an etiologic rôle and which are probably congenital in origin. (2) Acquired, true diverticula—those in which all layers are present although there may be thinning of the gastric layers with evidence that some disease was an etiologic factor. (*a*) pulsion type resulting from intragastric pressure; (*b*) traction type secondary to extragastric adhesions. (3) False diverticula—resulting from a break in the gastric wall secondary to disease.

According to this classification, penetrating ulcers both benign and malignant might fall into the third subdivision of false diverticula. This would appear only to confuse the issue. Furthermore, it is neither certain that all congenital diverticula present an intact replica of the gastric wall[14] nor that all acquired diverticula show a mural deficiency.[15] Martin[4] has collected fifteen cases of diverticula which showed islands of pancreatic tissue in the tip. These are considered to be of the congenital variety.

From the standpoint of the clinician a classification based on a rigid separation of congenital from acquired diverticula does not prove practical. This differentiation cannot be made by roentgenologic examination and in the final analysis, according to most definitions, diagnosis rests in the hands of the pathologist following microscopic examination. In order to eliminate the possible inclusion of penetrating ulcers in this category of gastric disorders a prerequisite for diagnosis of a diverticulum is that it be lined by gastric mucosa. Obviously ulceration may involve this lining secondarily; however, the mucosa will be found to dip into the out-pouchings in marked contrast to the histology observed in ulcer craters.

In the interests of simplicity and practicality a morphologic classification will probably prove more satisfactory than an etiologic one, especially since the etiology is so often questionable. The following classification is offered: (1) True diverticula in which all layers of the gastric wall are present. (2) False diverticula in which all or part of the muscular layers or serosa may be absent.

While most true diverticula are congenital and most false diverticula are acquired, there is no reason why pulsion or traction may not play an etiologic rôle in true diverticula nor why false diverticula may not be either congenital or acquired in origin.

ETIOLOGY

Formation of a gastric diverticulum is dependent on the existence of a "locus minoris resistentiae." This may be an intrinsic mural weakness, secondary to deficient or faulty muscle arrangement or it may be extrinsic in origin secondary to inflammatory contractures or increased luminal tension. Frequently a combination of the aforementioned factors completes the picture.

The cardiac region of the stomach is that most frequently involved by gastric diverticula[10] and it is in this region that one finds two contributing factors to the mural defects: first, a muscle deficiency and second, an abundant penetration by large vessels. The longitudinal muscular fibers of the stomach thin out as they approach the cardia until there are portions in which

the mucosa is supported only by a rather thin layer of circular fibers. A somewhat similar situation exists at the pharyngo-esophageal junction, another site for diverticula. Cunha[16] has aptly described this area of muscular deficiency in his lucid presentation of drawings and microscopic sections.

The coeliac axis lies in close proximity to the cardio-esophageal junction and numerous branches from the left gastric, inferior left phrenic and the splenic arteries pierce this portion of the stomach. It stands to reason and is borne out by actual observations made during surgical operations (two case reports presented by the authors) that these penetrating vessels might very well play an important rôle in the genesis of diverticula, not only in the formation of a mural hiatus but also in the fact that the vessels themselves act as traction factors.

Considerable credence is given the etiologic rôle of pulsion and traction by Martin[4] who classifies diverticula into these two types. If certain points of mural weakness associated with conditions in which there is increased intragastric tension or extragastric adhesive traction exist, the stage is set for possible development of a diverticulum. Cunha[16] validates this sequence in his case reports in which three patients had pyloric obstruction and two gave a history of pertussis with chronic severe cough. Jordan and Lahey[17] believe that perigastritis plays a rôle in formation of traction diverticula. Numerous case histories describe adhesions from the surrounding viscera and the abdominal wall which must be construed as playing an important part in the genesis of certain gastric diverticula.[2,3,12,18–21]

Location. Diverticula may be located anywhere in the stomach but the most frequent site is on the posterior wall of the cardia.[8,22–24] Martin[4] in his study of 103 cases found that 61 per cent of the diverticula were situated within a few cm. of the cardia. These will usually be found to lie on the posterior wall near the lesser curvature and within 3 cm. of the esophageal opening. The next most common site is adjacent to the pylorus, more frequently along the lesser than the greater curvature. Shiflett[25] in his series of forty-three collected cases reported 65 per cent to be located at the cardiac end and 23 per cent near the pylorus with the remainder involving various other parts of the stomach.

Incidence. Owing to the infrequency of this condition, most of the reports deal with single or small groups of cases which makes it rather difficult to collect accurate statistics as to incidence. Gastric diverticula, with the exception of the ileojejunal section, are the least frequent of all gastrointestinal out-pouchings. Shiflett[25] made 768 roentgenologic studies with the following incidence of diverticula: colon, 4.96 per cent; duodenum, 3.74 per cent; esophagus, 0.79 per cent; stomach, 0.65 per cent and only one case of a diverticulum of the small bowel. In 19,022 roentgenologic examinations Reich[24] found only six cases. Rivers et al.[3] found ten cases in 11,234 consecutive exploratory operations on the stomach and made twenty-five roentgenologic diagnoses of gastric diverticula in the course of 91,532 routine roentgenologic examinations of the stomach. Rigler and Ericksen[26] reported two cases in a series of 4,236 roentgenologic examinations of the stomach.

Shiflett[25] made an analysis of his series of forty-three collected cases as to sex and age incidence. He found males made up 44.2 per cent and females 55.8 per cent of the series. As to age periods there were in the first decade two patients, the second decade none, the third decade six patients, the fourth decade eleven patients, the fifth decade thirteen patients, the sixth decade nine patients and beyond this age there was one patient.

Pathology. The usual histology of the uncomplicated true diverticulum reveals all layers of the gastric wall to be continued, possibly with some thinning, throughout the out-pouching. In the case of false diverticulum one usually finds the gastric

mucosa and submucosa to be intact while the muscular layers will thin out and end abruptly near the border. The serosa is frequently absent in the presence of adhesions such as are found in the traction type of diverticula.

Martin[4] has collected a series of fourteen cases in which islands of pancreatic tissue were observed in the apex of the diverticula whose walls otherwise were made up of normal-appearing gastric layers. In all but one of these the location was at the pyloric end. Gile[27] presented a case of gastric diverticulum which showed typical colon mucosa on about one-half the wall and pyloric type of mucosa on the remainder. Rivers[3] reported a sarcoma in the wall of one patient and an adenomyoma in another. Mayo[28] and Mellon[29] each report a case of carcinoma in the wall of a gastric diverticulum.

Perusal of the literature fails to reveal many of the complications found more frequently in diverticula of other portions of the gastrointestinal tract. Acute perforation, frank diverticulitis and strangulation are not reported in the available literature. Hartley[30] reporting in the British Journal of Radiology presents a most intriguing set of x-rays showing a large gastric diverticulum in a left inguinal hernial sac.

SYMPTOMATOLOGY AND DIAGNOSIS

From the standpoint of symptomatology there is no particular or specific pattern which fits this clinical entity and anyone who has persistent epigastric distress must be considered as possibly having a gastric diverticulum. The usual sequence of events resulting in a diagnosis is a group of symptoms related to the gastrointestinal tract, a diagnostic barium meal and then, instead of the discovery of an ulcer or a muscular dysfunction, the investigator finds a diverticulum.

The next problem confronting the diagnostician is whether or not the newly discovered pathologic condition is the etiologic factor in the symptomatology. The diverticulum is usually asymptomatic and one must think twice before explaining symptoms upon the basis of demonstrating such a condition. Further examination may reveal a gastric or duodenal ulcer thus showing the diverticulum to be an incidental finding.

The diagnosis rests entirely with a careful roentgenographic examination. The sac, if located in the most common site at the cardiac end of the stomach, is visualized immediately after the barium enters the stomach. However, follow-up studies are most important. Retention of barium in the diverticulum after four hours indicates poor emptying of the sac and probably indicates that a symptomatic condition is present. It is true that in the differential diagnosis two conditions must be considered: first, perforated peptic ulcers and second, ulcerative carcinoma of the stomach. It is not the purpose of this paper to go into a detailed account of the differential diagnosis but if in doubt it may be necessary to resort to gastroscopic examination.[13]

TREATMENT

Medical treatment should be instituted before surgery is considered. This treatment consists of postural drainage, small frequent feedings and antispasmodics. It is true that many of the cases reported in the literature had neuropsychiatric symptoms and evaluation of such symptoms are extremely important before any surgical procedure is considered. If after six weeks of medical regimen and proper psychiatric evaluation the patient is still symptomatic, surgery should be contemplated. The surgical procedure, while reported by some authors as extremely difficult,[8] was carried out in our two patients without serious technical difficulty.

The approach to most gastric diverticula is best made by a midline incision. In certain cases of fundal diverticula a transthoracic approach may prove satisfactory.[11] In the two cases presented the diverticula was exposed by section through the gastrocolic and the gastrosplenic ligaments with rotation of the stomach to the right. Even

though the pathologic out-pouching lay closer to the lesser curvature, the approach was technically easier around the more flexible greater curvature. (Figs. 1 to 3.)

ing antiacid, antispasmodics, frequent feedings and the convalescent ulcer diet produced no relief of symptoms.

On February 8, 1946, surgery was performed

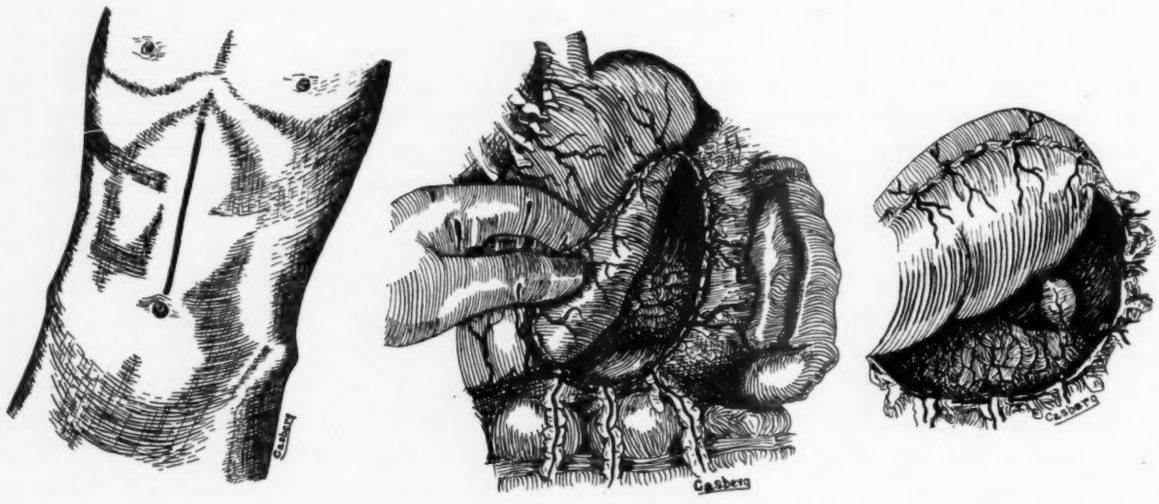

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Fig. 1. Midline xipho-umbilical incision which can be extended inferiorly or transversely should the need arise. In the exposure of certain parts of the fundus a transthoracic approach may be utilized.

Fig. 2. The greater curvature of the stomach being more mobile an approach through the gastrosplenic and gastrocolic ligaments with rotation of the stomach to the right proved more satisfactory than an approach by way of the lesser curvature and gastrohepatic ligament.

Fig. 3. A fairly large branch of the splenic artery was found running into the apex of the diverticula in both cases. This is of interest from the standpoint of etiology, both as a factor of intrinsic weakness in the gastric wall and also as an element of traction.

CASE REPORTS

Case 1. Mrs. W. K., aged forty-eight, a housewife, complained of intermittent burning and gnawing pains through the mid-chest during the preceding five years. She stated that occasionally she had the sensation of having had food stuck in her chest. There had been no weight loss. The pain was more severe when the patient lay on her left side and it was occasionally relieved to some degree when she lay on her right side.

Examination revealed a well developed, fairly well nourished, adult female in no acute distress. Complete physical examination revealed no abnormalities. X-ray study of the stomach demonstrated a diverticulum of the lesser curvature at the cardiac end of the stomach, posteriorly, immediately below the esophageal orifice. (Fig. 4.) At three and twenty-four hours after ingestion barium was still retained in this diverticulum. (Figs. 5 and 6.) Gastric analysis of fasting specimen revealed no free acid and a combined acidity of 14 degrees. The sedimentation rate was 12 mm. There was no anemia. Medical regimen included and a diverticulum was found in the posterior aspect of the stomach near the lesser curvature just distal to the esophagus. The diverticulum was attached to the posterior abdominal wall by a vascular pedicle which entered the pouch at its apex. The diverticulum was 2 cm. in diameter and had a large sessile base. It was removed and the postoperative course was uneventful. Pathologic report revealed the presence of gastric mucosa, muscularis mucosae and submucosa but no muscularis or serosa. There was extensive inflammatory reaction in the mucosa which consisted chiefly of lymphocytes. Follow-up x-ray of the stomach on March 29, 1946, revealed a normal stomach. (Fig. 7.)

The patient continued to complain of gastric distress following surgery and she was placed on a strict ulcer regimen. On the 29th of April it was found that she had gained weight and her general condition had improved. However, as yet her case cannot be classed as a complete cure because of the marked functional element involved which requires further treatment. There has, however, been definite improvement and the diverticulum has been removed.

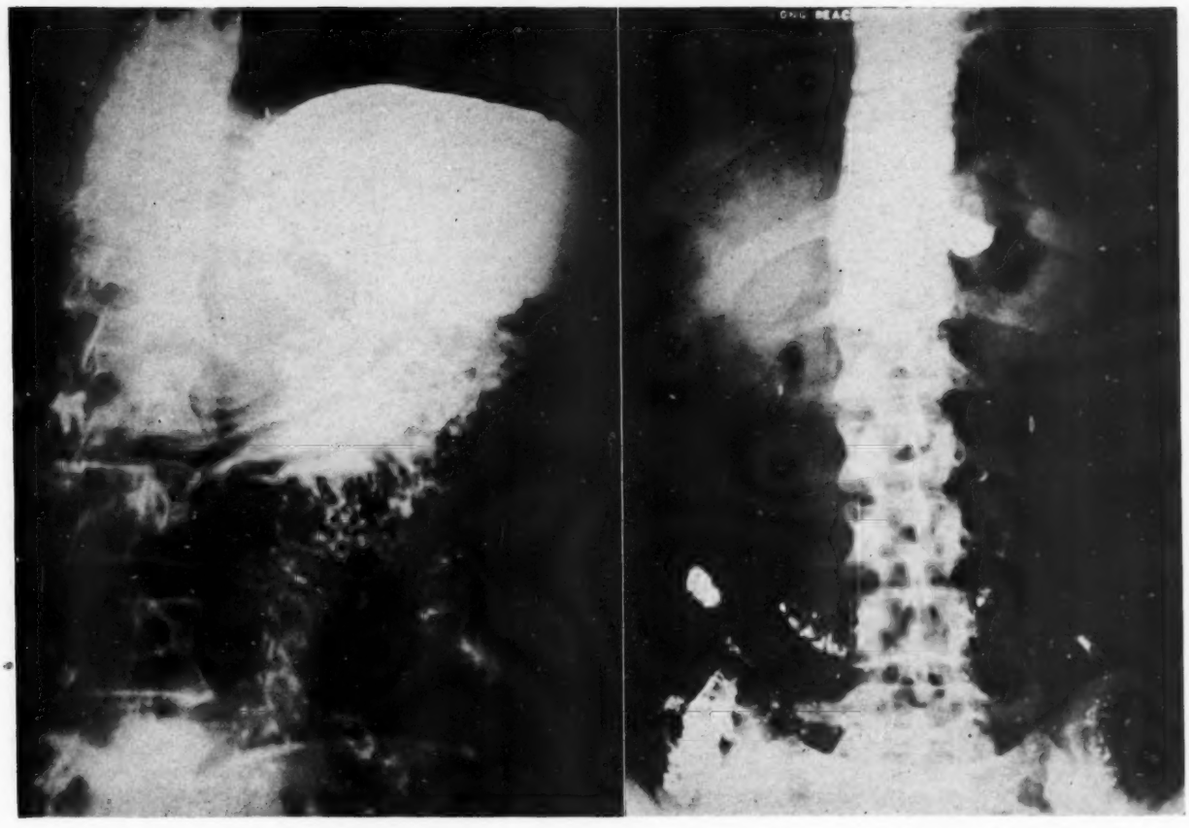

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Fig. 4. X-ray following barium meal in Case 1 showing a diverticulum at the cardiac end of the stomach posteriorly, immediately inferior to the esophageal orifice.
Fig. 5. X-ray in Case 1 three hours after ingestion of barium with diverticulum clearly outlined.

Case II. Miss M. R., aged nineteen, telephone operator, complained that during the last year she had had epigastric pain occurring one hour after meals. The pain was aggravated by cold drinks. There had been moderate anorexia and a loss of 10 pounds in weight during the past three months. Antiacid and ulcer regimen gave no relief. There was thought to be a strong functional element and the patient was advised that she should be careful to evaluate her symptoms and was assured that no definite benefit could be promised by surgery. Attempts were made to increase her weight by administration of amino acids and frequent high calory feedings but her symptoms persisted. The patient complained that she could not endure the nocturnal epigastric pain which caused her to remain awake most of the night and she insisted upon surgery.

Surgery was performed on March 29, 1946, and a large diverticulum was found in the posterior surface of the stomach near the lesser curvature about 1 inch below the esophageal orifice. The diverticulum was pedunculated, in that the opening into the stomach was narrow. Two large vessels, branches of the splenic arteries, penetrated the gastric wall in the region of the base of the diverticulum. The pathologic report revealed only the mucosa and submucosa and a moderate amount of chronic inflammatory reaction. The diagnosis was chronically inflamed gastric diverticulum.

Preoperative x-ray diagnosis revealed a diverticulum of the stomach on the posterior wall, about 2 cm. in diameter, located approximately 1 inch below the cardiac orifice. (Fig. 8.) Examination at four hours after the barium meal revealed retention in the diverticulum. (Fig. 9.) Incidentally, x-ray examination of the stomach done at another hospital on January 6, 1946, about six weeks before the patient was operated upon, failed to visualize this diverticulum although a later study of the films made at that time revealed evidence of the diverticulum. X-ray examination of the stomach six weeks after operation gave no evidence of gastric diverticulum. Postoperative x-rays gave evidence of some gastric retention indicating

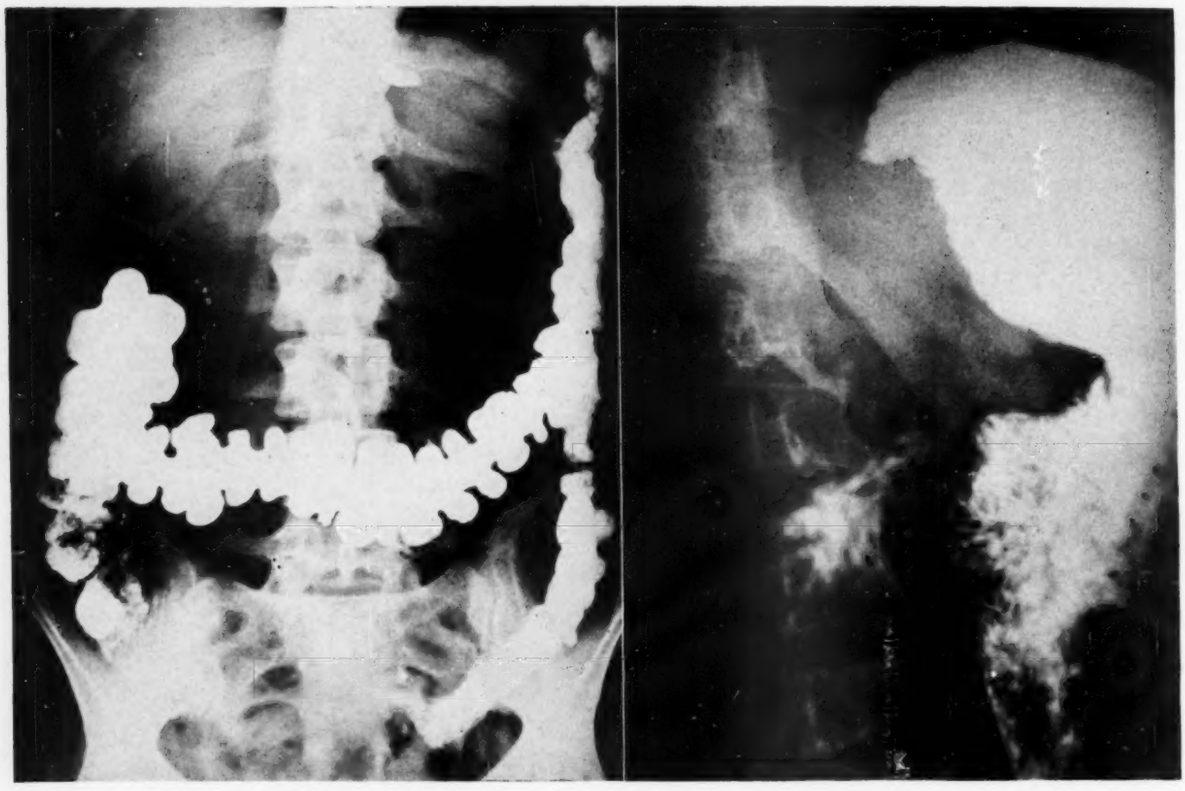

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Fig. 6. X-ray in Case 1 twenty-four hours after ingestion with barium still retained in the diverticulum.
Fig. 7. X-ray follow-up in Case 1 after surgery showing a normal stomach outline.

pyloric spasm. (Figs. 10 and 11.) After twenty-four hours the stomach was empty. The patient was given antispasmodics and a bland diet for about three weeks following surgery. This management proved to be very successful. There was no recurrence of nocturnal pain and the patient regained her lost weight. In a follow-up study it was found that there was no recurrence of the pain described in the original complaint although the functional element was still strong and reassurance was necessary to help the patient through the period of uncertainty which might have been expected in such a condition.

The results of all laboratory work, including complete blood count, urinalysis and sedimentation rate, were within normal limits preoperatively. Gastric analysis preoperatively revealed 20 degrees of free acid which was considered to be within normal limits after an alkaline meal.

A survey of the records at the Los Angeles County General Hospital for the period 1929 to 1945 revealed that six patients had been found to have gastric diverticula. The total admissions for this period were over 700,000. The figures indicate, therefore, that approximately .0008 per cent of all patients admitted during this sixteen-year period were found to have gastric diverticulum. However, there probably were some among these patients who had gastric diverticula but were without symptoms and thus it was not recognized. Of these six patients the following information was obtained:

Case 1: A case of a seventy-four year old white female who had had no clinical symptoms related to her digestive apparatus. Gastric diverticulum in the lower portion of the greater curvature of the stomach was found at autopsy. The patient died of congestive heart failure.

Case 2: This was a case of a fifty-four year old white female who gave no complaints relative to her digestive tract. On routine x-ray examination of the stomach she was found to have a diverticulum 1 cm. in diameter in the cardiac portion posterior

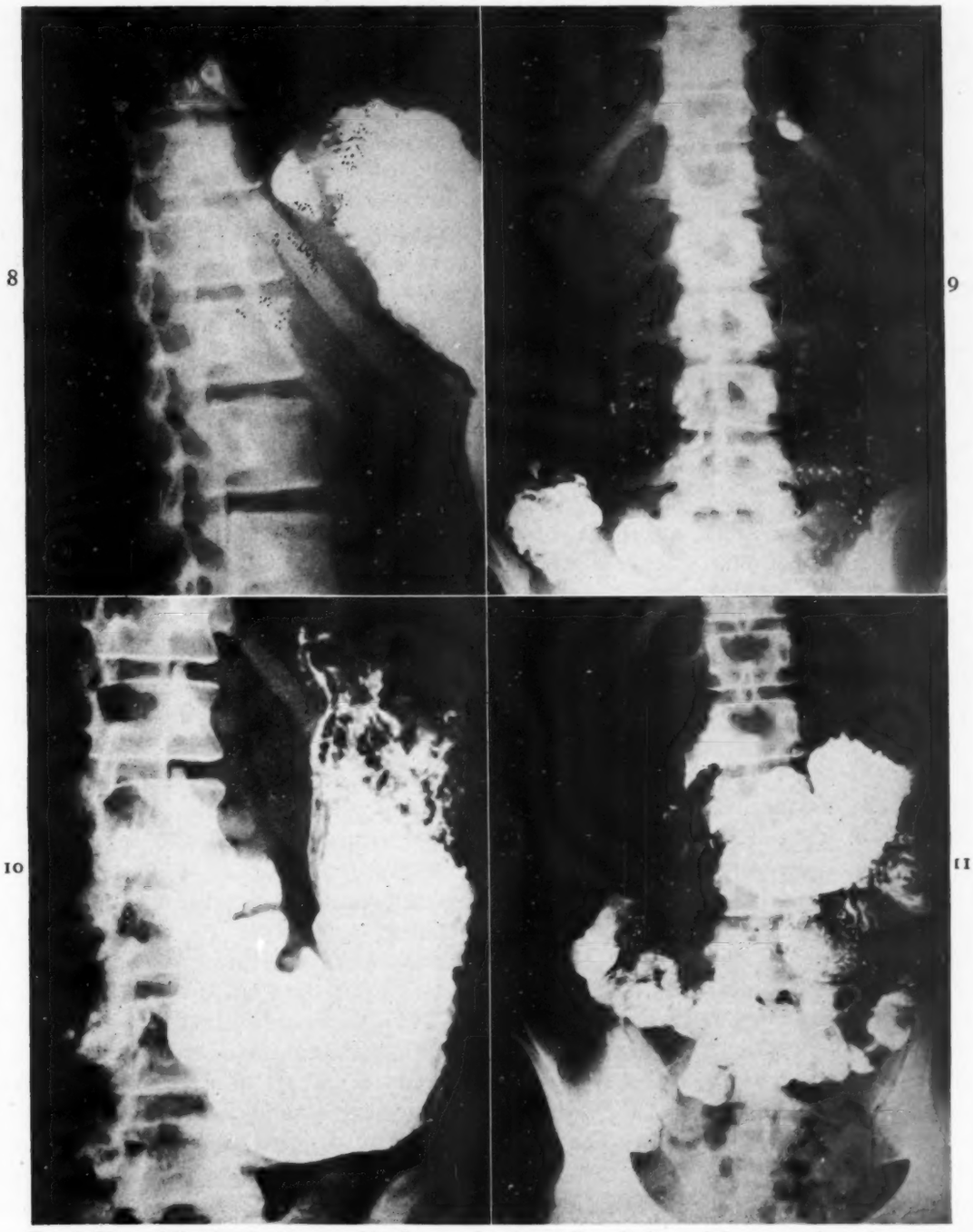

FIG. 8. X-ray following barium meal in Case II showing a diverticulum of the stomach at almost the identical location as in Case I.
FIG. 9. X-ray in Case II four hours after barium meal showing retention in the diverticulum.
FIG. 10. X-ray of the stomach in Case II six weeks after operation and immediately after barium ingestion showing normal outline.
FIG. 11. X-ray in Case II taken four hours after that in Figure 10 showing incomplete emptying of stomach.

to the esophageal opening. The patient was treated for congestive heart failure and ascites.

Case 3: This was a case of a forty-two year old white male who gave a history of excessive alcoholism. He had had acute abdominal pain and black stools for three days. X-ray examination revealed a diverticulum in the anterior aspect of the stomach attached to the fundus by a long pedicle. There was evidence of antral spasm. On conservative treatment the patient's condition improved and he was discharged.

Case 4: This patient was a fifty year old white female who gave a history of acute abdominal pain of three days' duration. X-ray examination gave evidence of a sacular dilatation of the proximal portion of the greater curvature at the pylorus. Gastric resection was performed. Microscopic study revealed a chronic, diffuse gastritis and atypical gastric mucosa in a diverticulum of the stomach. The patient expired about two weeks postoperatively. The diagnosis was postoperative diverticulum of the stomach, bronchopneumonia and syphilis of the central nervous system.

Case 5: The patient was a forty-four year old white female who complained of burning pain in the epigastrium and a loss of 25 pounds in weight during the preceding year. X-ray examination revealed a gastric diverticulum 2 by 3 cm. in diameter below the esophageal orifice. The patient was operated upon and the postoperative diagnosis was diverticulum and polyposis of the stomach. No follow-up studies were made.

Case 6: This was the case of a sixty-four year old white female who gave a history of nausea, epigastric distress and weight loss extending over a period of eight years. X-ray revealed a diverticulum in the superior posterior aspect of the stomach. Surgery was advised but was refused by the patient. No follow-up study was made.

SUMMARY

1. Gastric diverticula, with the exception of those in the ileojejunal section, are the least frequent of all gastrointestinal diverticula.

2. In the interests of simplicity and practicality a morphologic classification will prove more satisfactory than an etiologic one. (1) True diverticula in which all layers of the gastric wall are present and (2) false diverticula in which all or part of the muscular layers and perhaps serosa are absent.

3. Formation of a gastric diverticulum is dependent on the existence of a "locus minoris resistentiae." This may be an intrinsic mural weakness secondary to deficient or faulty muscle arrangement, or it may be extrinsic in origin secondary to inflammatory contractures or increased luminal tension or a combination of all or part of the aforementioned factors.

4. The most frequent site of gastric diverticula is on the posterior wall of the cardia.

5. There is no syndrome pattern which fits the clinical entity of gastric diverticula.

6. Diagnosis is usually made during the course of a gastrointestinal x-ray series in the process of searching for the cause of vague symptoms of epigastric distress.

7. As many gastric diverticula are asymptomatic one must not attribute existing symptoms to such a condition unless other possible gastrointestinal disorders have been ruled out.

8. Therapy should at first be conservative, including medical management and psychiatric evaluation. Should symptoms persist then one should resort to surgery.

9. The surgical approach used in the two cases herein presented was through a midline incision and around the greater curvature of the stomach. This afforded good exposure of the diverticula in both instances.

10. Two personal case histories are presented in detail as well as six cases from the Los Angeles County General Hospital.

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HEMORRHAGIC SHOCK

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THE purpose of this paper is to describe in some detail the problems of hemorrhagic shock as seen in the European Theater together with their method of handling and the lessons learned. The details outlined herein are a summation of the experience of the various surgeons and shock officers of an Auxiliary Surgical Group. The material has been culled from the personal files of the surgeons, official reports of the surgical team heads and from a monograph on acute traumatic surgery by the consultant surgeon for the Mediterranian-Italian Theater. Experiences are drawn from Tactical Operations of the Fifth and Seventh Armies. They commence with the beachhead operation of North Africa and end with the drive for central Germany. These experiences have for their background: (1) the airborne operation in the Low Countries, (2) Tunisian desert campaign, (3) various Italian river crossings and (4) battles associated with the Bulge and the Seigfried Line.

The shock officer is primarily concerned with the problems of shock and his duties are the supervision and treatment of all shock which occurs in battle casualties. This officer works in the most forward Field Hospitals where the patients treated are so critically wounded that they cannot be transported to the rear. Treatment of acute blood loss in these patients is the first responsibility of the shock officer. It is his duty to see that suitable blood replacement is carried out by blood transfusions and blood plasma. The amount and type of blood used or the use of blood derivatives are decisions which the shock officer has to make and are predicated on the clinical condition of the patient when he arrives in the shock tent.

Churchill states: "Restoration of blood volume and the relief of hypo-tension and anoxia must be done without loss of time. Replacement therapy forms the basis of resuscitation. Whole blood in large amounts is mandatory. One can speed resuscitation by simultaneous infusion of blood into one vein and plasma into another vein or into the sternal medullary cavity."[1] The important thing to remember, however, is that whole blood must be rapidly infused into the circulatory system even under pressure if the peripheral vasomotor collapse is so profound as to prevent the normal operation of the classical Baxter sets which we employed routinely. (By rapidly we mean 500 to 1,500 cc. in twenty minutes). Unfortunately, the idea has developed that the treatment of shock rests upon the restoration of blood volume. This is false reasoning. "The dominant factor in shock is the loss of *blood* into the wounded tissues, into body cavities, and to the surface. The successful treatment of shock, therefore, not only rests upon the restoration of blood volume, but also upon the restoration of hemoglobin as well. Infusions of plasma alone will not suffice. Beyond a certain point, plasma may do harm by diluting the remaining red blood cells at a time that the rising blood pressure increases hemorrhage. A false sense of security may be created by the elevation of the systolic blood pressure under these circumstances. A blood pressure brought to normal or nearly normal levels by plasma alone, may fail precipitously with the induction of anesthesia, operative manipulation, or merely moving the patient."[1,2]

The preparation of the critically injured battle casualty for surgery requires the utmost in keen surgical judgment and astute surgical diagnosis. Appraisal of the patient's condition demands the sum total of the surgeon's clinical knowledge. So, too,

the shock officer must be ever alert and mentally active for it is he who will care for the patient during the surgeon's absence or until such time as the surgeon can render his opinion. Reactivation of hemorrhage may occur as the circulation improves. If continuing hemorrhage is suspected, the surgeon must proceed with the indicated operation while blood is being transfused into one or more veins. If the state of shock does not improve and/or respond rapidly to infusion of blood and plasma, it is a fair assumption that there is continuing hemorrhage, established infection or that the continuing shock is associated with other injuries, for example, intracranial damage, transection of the spinal cord or open wounds of the thoracic cavity with the resultant disturbance of the cardiorespiratory physiology. One of our problems of shock is: When is a patient ready for surgery? There is no specific answer. Each case is a problem in itself, but the shock officer must hold the key to the situation. His duty is to save life; it is also his duty to restore a patient to a state of equilibrium which will permit the necessary surgical procedures. The shock officer outlines and carries out an individualized plan of shock therapy for each critically injured battle casuality admitted to his tent.

Of all the drugs and procedures developed for the treatment of hemorrhagic shock, none has stood up under the critical test of forward surgery with the exception of replacement therapy. External application of heat and the use of morphine are two time-honored measures in the treatment of shock which, if used injudiciously, could cloud the picture considerably. "The external application of heat can be dangerous if overdone."[2] Fluid loss in the desert campaign and the drive for Rome was great even in the normal man. What would it be like in a man with dysfunction of the vasomotor system due to shock? Morphine is advisable only in moderate amounts and its use is limited to the excited patient.

"Morphine is not indicated for the treatment of restlessness of hemorrhage, which is largely due to anoxia."[2] The "q.s. ad libitum" prescription for morphine can lead only to dire end results in two types of patients: (1) the patient with acute hemorrhagic shock and (2) the patient with an invasive anaerobic cellulitis infection.

The patient with acute hemorrhagic shock has no functioning peripheral vascular system and consequently no functioning route of assimilation. Morphine given to this type of patient by the usual route of administration does nothing more than pile up in the tissues adjacent to the site of injection. This produces a pernicious situation because there is delayed absorption of the drug. When the peripheral circulation is improved by resuscitation therapy, a manifold absorption of the piled up morphine ensues with resultant acute morphine poisoning.

A patient with acute invasive anaerobic cellulitis will have his diagnostic findings masked if injudicious morphine medication is given him. "The diagnosis of clostridial myositis in a field hospital is entirely clinical and is based in great part on minute alterations of the patient's psychic pattern."[3] Clostridium Welchii infections make for the apprehensive, hyperexcitable, occasionally maniacal type of personality change, whereas infections caused by Cl. perfringens and oedematiens project the mental pattern into the comatose category. In a case of clostridial myositis the mental base line must be established. Morphine medication militates against such a procedure and makes the diagnostic estimate most difficult.

We know that morphine is a good drug but it is also a two-edged sword. In the types of patients which we have described, morphine is indicated for definite and unequivocal pain only, and the route of administration should be intravenous to avoid additive effect. Use the drug to the limit but use it wisely and judiciously. The differential diagnosis between acute fulminating clostridial oedematiens infec-

tion and morphinism is not easy; the treatment of acute hemorrhagic shock complicated by morphine poisoning is not a simple task.

TYPICAL CASES OF SHOCK

Head and Face Injuries. The average head injury, if the patient lives for six hours, usually does not present much of a problem. In most cases the injury is such that the patient decompresses himself. The problem with head injuries is not as a rule too difficult to solve because the shock is not primarily due to acute blood loss unless there is an associated transection of one of the major vascular bundles. From the point of view of the shock officer straight forward head injuries are best managed by establishing an adequate airway and supplying hypertonic fluids. "Twice concentrated plasma" and serum albumin are the usual agents available in front line hospitals for this form of therapy. The establishment of an airway, especially in patients with associated facial injuries, resolves itself into performing a tracheotomy. If there is any doubt as to the embarrassment of the airway in a facial injury, it should manditorially point to a tracheotomy. We always believed that adequate oxygenation was a very important phase of shock therapy and failure to maintain adequate oxygenation was to sign and seal the death warrant of the patient involved.

Chest Injuries. In chest wounds the important problem is the restoration of the normal physiology of the cardiorespiratory system. Shock treatment in this type of injury is based upon the alleviation of the asphyxia, control of the pulmonary edema and the negation of tension pneumothorax. Actually, surgery in chest wounds is a secondary consideration. The primary consideration is shock due to altered respiratory function. The treatment of this special form of shock is immediate and rapid aspiration of the pneumothorax, closure of the perforation if there be one, control of pain by intercostal block, drying out of the tracheobronchial tree by catheterization and nasal oxygen medication and the prompt release of tension pneumothorax through a water seal. Finally, the crux of shock therapy in chest injuries is adequate stabilization. The knowing when to operate on a chest injury is just as important as knowing how. One must answer these questions: Is that portion of the functioning lung field dry? Has the patient's hemoglobin been restored to reasonably normal levels? Can the patient withstand additional asphyxia in the form of anesthesia?

Abdominal Injuries. Penetrating wounds of the abdomen will test the shock officer's diagnostic acumen to the nth degree. Patients with traumatic, intra-abdominal wounds have a most unstable vasomotor system. While their shock may well be ascribed to hemorrhage, one must keep in mind that irritative peritoneal contamination in the form of feces, bile, blood and urine alone cause profound shock and death. This untoward reaction is most often seen after injuries to right colon, ileum, and cecum whose contents are notably irritative. The heat and fluid loss of intestinal eviscerations and the acute pain of stomach avulsions are also major contributory factors in the cause of shock in abdominal injuries. The treatment of these patients is perforce blood. How much blood one will give is predicated upon several factors: (1) rise in blood pressure, (2) stability of blood pressure, (3) hematocrit and plasma protein readings as determined by the Evans copper sulfate method, (4) clinical appearance of the patient and (5) the tactical situation with its associated operating load.

In addition to blood there are other therapeutic agents which are essential in the treatment of shock in abdominal injuries. The stomach should be decompressed as soon as the patient arrives in the shock tent. The reasons for the use of a gastric suction tube in gut injuries are obvious. The employment of the gastric suction tube in other conditions is also of value. In pregnancy the processes of

gastric motility cease with the onset of labor. So, too, when a man is critically injured, his gastric functions temporarily cease. Immediately acute gastric dilatation sets in. This causes pain but it also raises the diaphragm on the left side and thereby reduces pulmonary ventilation. The reduction of available aeration space in the lung is the one thing one does not want in shock; certainly it is an undesirable factor in thoracic, abdominal and thoraco-abdominal wounds.

Another factor which will reduce vital capacity is the voluntary splinting of the abdominal musculature and its concomitant decrease of the costal excursion. Penetrating wounds of the upper abdomen notoriously develop wet lung and it is our opinion that part of this may be attributed to this abdominal splinting. The well trained shock officer will be thinking about muscular splinting at all times. Sensation of the abdominal wall arises from the ninth thoracic through to the twelfth. Treatment of upper abdominal wounds, in part, should be directed toward blocking the previously mentioned innervation. It is in this fashion only that the patient can be prevented from reducing his vital capacity by muscular splinting of the abdominal wall. A shock officer has only to treat the pulmonary edema which arises from poor ventilation once; he will ever after keep this important matter in mind.

Extremity Injuries. The cause of shock in straight forward fracture cases seen in civilian life has never been too well accepted by all. Various theories have been advanced, all of which are basically sound. But the cause of shock in extremity wounds due to shell fire and mines is obvious. These patients manifest such pathologic conditions as fracture of the long bones with and without extensive comminution and/or soft tissue loss, major vascular injuries complicated by hemorrhage, traumatic amputations both partial and complete, penetrating wounds of the joints, transections of the major nerves and penetrating wounds of the thighs with associated perforations of the bladder, rectum and peritoneal cavity. A complication not infrequently seen in extremity wounds is anaerobic cellulitis both invasive and non-invasive. Any one or a combination of these factors will produce shock of a profound degree. The problem for the shock officer in evaluating a given case is to determine what factor or factors are producing the shock.

The loss of blood from a lacerated artery is apparent, but the loss of blood from venous back pressure due to an improperly applied tourniquet is often without reckoning. Frequently patients have been exsanguinated because of a tourniquet sufficiently tight to occlude the venous return but not the arterial supply. This is not the fault of the tourniquet but rather the method by which it was applied. The shock officer must be ever watchful of this procedure because it does happen more often than one realizes and it can be not a little embarrassing. The use of tourniquets in any case is very limited. The good shock officer can control all hemorrhage in extremity wounds either by direct pressure over the wound or by ligature of the offending vessel. If a tourniquet must be applied, it should be applied as low as possible. There are good reasons for this remark. In the first place, why compromise the vitality of the entire leg by placing a tourniquet over the femoral trigon when the patient has a traumatic amputation through the ankle. To compromise the vitality of an extremity in this fashion not only reduces the prospects of getting a good operative result but it also invites the development of anaerobic cellulitis in tissue which would otherwise be healthy had its blood supply not been impaired. Another reason for placing the tourniquet so low is that we wish to prevent the dissemination of myoglobin and exotoxins of clostridia from the local site of trauma.

Extremity wounds present another situation which at times can be most confusing. This is infection with anaerobic organisms. Anaerobic infections are important to the

shock officer because they are so often the etiologic basis for the patient's shock. Patients with extremity wounds can and do go into shock three hours after wounding and a frequent cause is acute invasive anaerobic cellulitis. Unfortunately, the diagnosis of such conditions is entirely clinical. Were we to see infections due primarily to Clostridium Welchii and Clostridium perfrigens, the matter would be relatively simple. The crux of the argument is that infection caused by these organisms is not always the common finding. "Intensive studies carried out in Sicily, Italy, and France disclosed that Cl. Bi-fermentens, Oedematiens, and Sordelli infections were also frequent offenders."[3] It was the toxemia elaborated by these organisms which threw the patient into decisive, profound and rapidly fatal shock. The existence of these organisms with their fulminating type of infection should be borne in mind at all times because their diagnosis all too often hangs by the fine thread of an unstable blood pressure, a rapid pulse, minute changes in the general condition with alterations in the psychic pattern and mental behavior.

UNFAVORABLE RESULTS

The shock officer will have many disappointments no matter how astute he might be or how hard he works. He will have many mistakes written to his credit; his work by definition will make for a high mortality rate. The author in an attempt to learn wherein he had failed spent several months in the Pathology Department of a medical laboratory studying the postmortem findings of his errors.

When the protocols of the various cases were studied, and this includes many of those patients who were operated upon by surgical teams of the auxiliary surgical group but who never fully recovered from shock, two contributory causes of death were noted: (1) fat embolism and (2) hemoglobinuric nephropathy.

"There were two significant points noted in cases of fat embolism: (1) It was seen in cases who had severe fractures and/or traumatic amputations and (2) it was rarely diagnosed clinically."[4] In the medical laboratory files there were records of approximately 200 patients who died from wounds in which major fractures were a predominating complication. Analysis of the tissue slides of these cases showed thirty of them to have fat emboli and this condition was apparently not recognized clinically. The author is not without guilt in this matter. He had two patients who died presumably of clostridial myositis. Microscopic examination of the tissues taken from these two revealed the diagnosis to be in reality fat embolism.

As further study of the autopsy protocols was carried out, it appeared that fat embolism had its origin in four types of conditions: (1) traumatic amputations, (2) fractures, (3) extensive tissue injuries to buttocks and (4) burns.

The clinical picture of fat embolism is highly imitative. Characteristically, the condition seemed to manifest itself forty-eight hours after operation or major manipulative procedure. But we often saw patients with severe comminuted fractures of the forearm who would arrive in the shock tent four hours after injury and then promptly die for no apparent cause. The mode of demise in these cases was "wet lung"; the cause of the wet lung—pulmonary fat embolism. In those patients who manifested their symptoms forty-eight hours after operation one saw initially respiratory embarrassment usually dyspnea, cyanosis and ultimately frank pulmonary edema. Further findings were those of a cerebral nature such as mental confusion, hyperexcitability, amnesia, aphasic phenomena and sometimes convulsions and coma. Various clinically unexplained localizing signs presented themselves. The clinician's diagnostic acumen was severely tested when such conditions as retinal hemorrhage or edema, motor paralysis and disturbance of the vegetative centers in the form of hyperthermia became apparent. Manifestations of this sort can be confused

with possible head injury and indeed they often were. One patient was diagnosed as a Waterhouse-Friedrichson's syndrome and another case was written up as "Typhus fever, acute severe." According to the histories both of these patients manifested mental confusion, hyperthermia, shock, petechial hemorrhages in the skin and meningismus. These were logical conclusions. Pathologically, the story was a different matter. The slides showed fat emboli in the brain, kidney, lungs and connective tissue.

Another contributory cause of death in several of these cases studied was hemoglobinuric nephropathy. Hemoglobinuric nephropathy is the sword of Damocles in those patients receiving massive transfusions (5,000 to 6,000 cc.). Hemoglobinuric nephropathy (shock kidney—transfusion kidney—kidney of crush syndrome) is a degenerative and proliferative type of lesion. The first thing seen in the microscopic section of the kidney are the pigmented casts located in the tubules. With formation of this cast an obstruction and a concomitant dilatation of the proximal nephrons develop. Edema of the interstitial tissue ensues and one sees connective tissue stroma infiltration with histiocytes, lymphocytes and eosinophiles. The tubular epithelium, as a compensatory measure for the blocking, proliferates occasionally to the extent of granuloma formation.

The casts are derived from one of two substances: (1) hemoglobin and (2) myoglobin. On the microscopic slide both of these substances have the same chemical characteristics. To differentiate them one has to resort to spectroscopic procedures.

From the surgical point of view, hemoglobin, as seen in hemoglobinuric nephropathy, can be derived from three sources: (1) Transfusion of blood which is frankly incompatible, which may be due to mismatching or to the Rh factors which manifest themselves late (one to two months) and after many transfusions; (2) transfusion of hemolyzed blood, which has been stored blood past its expiration date or has been poorly prepared and/or cared for in the blood bank. (3) transfusions of *large amounts* of type "O" blood in a *short time.* The factor here is that with rapid transfusions hemolysis of the recipient's red blood cells takes place by A and B agglutinins contained even in low titer "O" blood. In other words, the protective factor of dilution, neutralization and excretion are negated.

SUMMARY

The dominant factor in shock is a loss of blood. The successful treatment of shock is predicated upon the rapid restoration of the normal hemoglobin content.

To have a thoroughly dry, patent, tracheobronchial tree with fully aerated, freely expansile, lung tissue is axiomatic. Tracheotomy, bronchial catherterization, intercostal blocks and oxygen medication are adjuvant procedures to which you must resort if you will treat shock successfully.

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MECKEL'S DIVERTICULUM

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THIS review covers the experience of the Rhode Island Hospital with Meckel's diverticulum and its complications during a twelve-year period beginning January 1, 1935 and ending December 31, 1946. The purpose of the review is twofold: First, several of the cases are of unusual interest; second, the review as a whole provides a means of self-analysis and self-criticism. It is our opinion that the Rhode Island Hospital represents an average, general hospital in a non-teaching center. For this reason our reflections, conclusions and experiences may benefit or at least interest similar institutions.

HISTORIC AND EMBRYOLOGIC BACKGROUND

This aspect of the subject has been adequately treated in many publications.[1,2,3] The embryologic facts of pertinent interest may be briefly summarized: The omphalomesenteric duct, which connects the yolk sac with the intestine, begins to be obliterated in about the fifth week of fetal life. Obliteration is complete at about the seventh week. Various degrees of failure of obliteration lead to various disease entities. First, if the entire duct remains patent, a fecal fistula opening at the umbilicus results. Second, when the distal end fails to close, intestinal mucosa is seen at the umbilicus where the secretions of the glandular elements form a discharge. Third, a retention cyst is formed if the mid-section persists. Fourth, persistence of the proximal end may be seen as an outpouching of the intestinal tract usually of the terminal ileum. This is what is commonly referred to as Meckel's diverticulum. It differs from other diverticula in that it contains all the layers of the muscularis of the intestinal wall. Occasionally associated with it is persistence of a fibrous stalk connecting its tip with the inner aspect of the anterior abdominal wall in the region of the umbilicus. This is the result of incomplete regression of the omphalomesenteric duct. The blood supply of the diverticulum usually comes from its mesentery, which arises from the mesentery of the bowel, or it may come from the bowel wall itself in some instances.

GENERAL CONSIDERATIONS

Site of Diverticulum. Meckel's diverticulum may arise from any portion of the intestinal tract although the terminal ileum is the usual site; it may arise from any point in the circumference of the bowel, including that area between the leaves of the mesentery. The usual point of origin is the anti-mesenteric border. In this series the extremes were a diverticulum arising only ½ inch from the ileocecal valve and one arising 2 feet proximal to the valve.

Size. The size varies greatly. The largest recorded in the literature is 104 cm.[4] although an average diverticulum is about the size of a thumb, no unusually large diverticula were encountered in this series.

Incidence and other Statistics. Figures derived from autopsy material show an incidence ranging from 1 to 2.5 per cent of patients coming to autopsy.[2,3,6] Various series show that the condition is more common in males than in females, the ratio ranging from 2.1 to 4.1.[3] In the author's series the ratio is three males to two females.

It is of interest to note the percentage of patients in whom Meckel's diverticulum was discovered and which was responsible for the symptoms. This figure varies from 20 to 35 per cent in other series.[4,5,7] In ours it is 42 per cent.

The age incidence in this series parallels that of other series. A notable finding in

relation to age incidence is that the percentage of patients showing a pathologic condition of the diverticulum is higher in the first two decades. Twenty-two of our cases occurred in the first two decades; fifteen of these, or 68 per cent showed

AGE INCIDENCE

Age (Years)	No.	No. Showing Pathologic Changes
Less than 2	5	3
2–10	8	6
11–20	9	4
21–30	13	5
31–40	7	0
41–50	3	1
51–60	5	2
Over 60	0	0
Totals	50	21

pathologic changes, whereas only nine, or 32 per cent, of twenty-eight cases occurring in patients after the second decade showed pathologic changes.

CLASSIFICATION

Greenblatt's classification[8] of disorders of the omphalomesenteric duct is one of the most adequate and inclusive. We have modified it to a certain extent by removing the heterotopic cases from the tumor group and classifying them as a separate group in which we include cases of peptic ulcer which Greenblatt has classified separately. The revised classification follows:

1. Obstructive Group 11
   - (*a*) Intussusception 2
   - (*b*) Volvulus 4
   - (*c*) Bands and adhesions 3
   - (*d*) Contents of hernia 2
2. Diverticulitis Group 5
   - (*a*) Simple acute inflammation 3
   - (*b*) Acute with perforation and gangrene 0
   - (*c*) Chronic inflammation 1
3. Heterotopic Group 10
   - (*a*) Gastric mucosa 7
     - (1) with ulcer and hemorrhage 1
     - (2) with ulcer and perforation 4
     - (3) with ulceration 0
     - (4) without ulceration 2
   - (*b*) Other tissues
     - (1) pancreas 2
     - (2) duodenum 0
     - (3) colon 1
4. Umbilical Group 0
   - (*a*) Fecal fistula 0
   - (*b*) Umbilical adenoma 0
5. Tumor Group 0
   - (*a*) Malignant 0
   - (*b*) Benign 0
6. Incidental Group 29

Figures in the right hand column show the number of cases in this series falling into each group. One of our own cases remains unclassified. None of the cases in our series fell into groups 4 and 5 or into 2(*b*).

Obstructive Group. Eleven of our cases are included in this group. (Table 1.) There were two cases of intussusception (cases 6 and 23). In both cases invaginated Meckels' diverticulum was the leading point of the intussusception. It is interesting that each of these diverticula contained a sizeable nodule of pancreatic tissue and these were the only cases in which pancreatic tissue was found. It seems possible that peristaltic movements of the diverticulum in attempting to expel its contents or an intrinsic mass could be responsible for invagination. Such a chain of events is suggested by Atwood in a recent paper.[9] Hunt and Bonesteel list four such cases in their compilation.[11] Ladd and Gross[10] report that of 372 cases of intussusception occurring in children fourteen were associated with Meckel's diverticulum. Another case of intussusception occurred in this series but there was no evidence that the diverticulum in this case was more than an incidental finding.

There were four cases of volvulus (Cases 1, 15, 20 and 33). In the last three a stalk was present running from the tip of the diverticulum to the inner surface of the abdominal wall in the region of the umbilicus about which loops of small bowel had become twisted. Figure 1 shows the diverticulum and stalk in Case 20. In case 15 the entire small intestine was twisted about the band. In Case 1 the volvulus involved a two-foot loop of terminal ileum. In the mid-point of this loop was an inflamed Meckel's diverticulum but it cannot be ascertained from the operative

FIG. 1. Meckel's diverticulum can be seen attached to the under surface of the umbilicus; X, umbilicus; A, B and C, Meckel's diverticulum; X—Y, ileum.

note whether or not the diverticulum was an integral part of the disorder. (Figs. 2, 3 and 4.)

Disturbances as the result of bands and adhesions are illustrated by Cases 8, 24 and 25. Case 8 is of interest in that the patient showed signs of obstruction eight days after the removal of an acute appendix and operation showed a Meckel's diverticulum looped over the ileum and compressing it. Two other points of obstruction, the result of adhesions, were freed and the diverticulum was left in place because of the patient's poor condition. Three months later the patient returned to the hospital complaining of frequent crampy periumbilical pains with nausea and vomiting. An operation revealed distention above the point of origin of the diverticulum. Adhesions so distorted and kinked the ileum that obstruction had resulted. Resection of the loop bearing the diverticulum was then carried out. In the other two cases the position of the diverticulum so rolled and kinked the ileum that there was narrowing of the lumen at the point of origin with symptoms of intermittent partial obstruction.

In three cases a Meckel's diverticulum was found in a hernia. In one case it was an incidental finding but in Case 5 the patient had symptoms consistent with strangulation and a large, infarcted Meckel's diverticulum was the sole content of the hernial sac. In Case 43 an omphalocele was noted at birth and an operation carried out before the child was four hours old. It was found that adhesion of a Meckel's diverticulum to the wall of the cord prevented retraction of a loop of the ileum. The diverticulum was removed and the bowel restored.

Diverticulitis Group. Meckel's diverticulum is subject to inflammatory changes in much the same manner as the appendix but with this difference: The lumen and ostium of the diverticulum is usually greater than that of the appendix and obstructive types of inflammation are less frequent. Cases 16, 22 and 28 represent acute inflammation. Each had an abrupt, progressive history indistinguishable from appendicitis. One man had previously had his appendix removed and the diagnosis of acute diverticulitis of Meckel's diverticulum was made preoperatively. Chronic diverticulitis as a diagnosis is about as unsatisfactory as the diagnosis of chronic appendicitis. Case 27 showed pathologic changes consistent with this diagnosis. The patient had had the history and physical signs usually associated with acute appen-

TABLE 1
OBSTRUCTIVE GROUP

Case No.	Type of Adhesions	Age	Symptoms	Signs	Operative Findings	Pathologic Findings	Rx.
8	Ir tussusception	15	Periumbilical pain with nausea and vomiting	Right lower quadrant spasm and tenderness	Ileo-ileal intussusception with Meckel's leading	Venous infarction; pancreatic tissue	Loop with Meckel's diverticulum resected
23	Intussusception	5 mo.	Vomiting 24 hr.	Melenae, visible peristalsis	Ileocolic intussusception with Meckel's leading	Venous infarction; pancreatic tissue	Meckel's diverticulum resected
1	Volvulus	22	Lower abdominal cramps with nausea and vomiting 24 hr.	Right lower quadrant spasm and tenderness	Volvulus of 2 ft. loop of ileum with gangrene	Venous infarction	Loop resected
15	Volvulus	10	Periumbilical cramps with vomiting 2 days	Right lower quadrant and periumbilical tenderness and spasm	Entire small bowel formed volvulus about stalk of Meckel's	Tissue lost	Meckel's diverticulum resected
20	Volvulus	29	Periumbilical cramps and vomiting 24 hr.; attack 3 yr. before	Generalized spasm and tenderness; absent arisstalsis	Volvulus of terminal ileum about Meckel's and its stalk	Acute inflammation of Meckel's diverticulum	Meckel's diverticulum resected
33	Volvulus	9	Periumbilical pain and vomiting; 2 previous attacks	Right lower quadrant spasm and tenderness	Ileum twisted about band running from Meckel's to umbilicus	Chronic inflammation of Meckel's diverticulum	Meckel's diverticulum resected
8	Bands and adhesions	19	Periumbilical pain and vomiting 4 days; 2 previous operations	Negative	Bowel at base of Meckel's kinked and rolled on mesentery; lumen small	Peridiverticulitis of Meckel's diverticulum	Loop with Meckel's diverticulum resected
24	Bands and adhesions	26	Episodes of right lower quadrant pain and nausea 1 mo.	Right lower quadrant tenderness and spasm	Tip of Meckel's held down by fibrous band and ileum kinked	Meckel's diverticulum	Meckel's diverticulum resected
25	Bands and adhesions	27	Episodes of epigastric pain, obstipation and nausea 5 yr.	Tenderness and spasm to right of umbilicus	Arose from lateral aspect of ileum; ileum kinked and lumen narrowed	Meckel's diverticulum	Meckel's diverticulum and loop of ileum resected
5	Contents of hernia	54	Incarcerated hernia	Irreducible tender mass; inguinal region	Red swollen Meckel's sole contents of herniae sac	Venous infarction of Meckel's diverticulum	Meckel's diverticulum resected
43	Contents of omphalocele	3 hrs.	Omphalocele noted at birth	Omphalocele noted at birth	Meckel's adherent to wall of sac, prevented retraction of ileum	Tissue lost	Meckel's diverticulum resected

2

3

FIG. 2. A plain film of the abdomen showing the presence of dilated loops of small intestine which led to the making of serial studies of the small intestine.

FIG. 3. Meckel's diverticulum is present at the constricted area. The roentgenologist believed that this represented a retroperitoneal tumor which was encroaching on the lumen of the terminal ileum. Note the dilatation of the proximal loop.

TABLE II

DIVERTICULITIS

Case No.	Age	Symptoms	Signs	Operative Findings	Pathologic Findings	Rx.
Simple Acute Inflammation						
16	52	No history recorded	No history recorded	Red, angry Meckel's diverticulum 4.2 cm. by 1.2 cm.	Acute inflammation	Meckel's diverticulum resected
22	41	Previous appendectomy; severe right lower quadrant pain with nausea and vomiting 24 hr.	Spasm and tenderness in right lower quadrant; tender on right on rectal examination	Inflamed diverticulum Meckel's 4½ inches long	Acute inflammation	Meckel's diverticulum resected
28	9	Upper abdominal pain shifting to right lower quadrant 24 hr	Spasm and tenderness in right lower quadrant	Inflamed Meckel's diverticulum 1.5 cm. long	Acute inflammation	Meckel's diverticulum resected
Chronic Inflammation						
27	16	Right lower quadrant pain with nausea and vomiting 12 hr.	Spasm and tenderness in right lower quadrant; tender on right on rectal examination	Large Meckel's diverticulum packed with feces	Chronic inflammation	Meckel's diverticulum resected

dicitis. A Meckel's diverticulum was found to be solidly packed with hard fecal material. (Table II.)

Heterotopic Group. Gastric mucosa, jejunal, duodenal or colonic mucosa and pancreatic tissue may be found in Meckel's diverticulum. Gastric mucosa is by far the most frequently found of those mentioned and is the only type consistently associated with symptoms. It is present in 16 per cent of all cases with Meckel's diverticulum according to Schaetz.[2] In children heterotopic gastric mucosa in the most frequent cause of symptoms; ulcer is the most frequent complication resulting from this disorder. Cobb[2] has reported a series of 110 cases showing ulceration in Meckel's diverticulum associated with the presence of gastric mucosa. The ulcer usually occurred in the immediately adjacent ileal mucosa, that is, near the base of the diverticulum. In Cobb's series hemorrhage occurred in 72 per cent and perforation in 55 per cent; 74 per cent of these patients were under the age of fifteen. No statistics are to be found which illustrate the percentage of those patients having gastric mucosa in a diverticulum who also have peptic ulceration. In addition to hemorrhage and ulceration a third complication is reported by Waugh et al.[12] These authors describe two patients in whom the scarring, resulting from the ulceration, so reduced the lumen of the adjacent bowel that obstruction resulted.

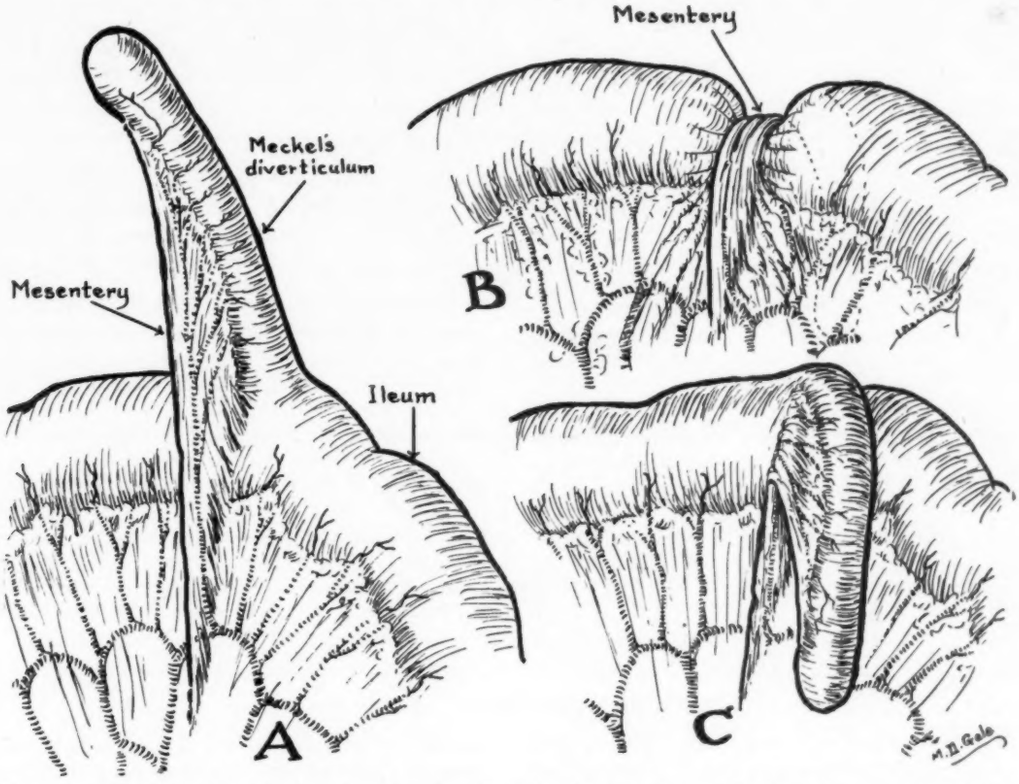

FIG. 4. A, Meckel's diverticulum hanging free in abdominal cavity; B, Meckel's diverticulum falling from the site of origin of its mesentery; note obstruction; C, Meckel's diverticulum falling toward side of origin of its mesentery; no obstruction.

The mechanism of ulceration is discussed by Cobb[2] and may be briefly summarized: Acid secretion of the gastric mucosa of the diverticulum parallels that of the stomach; it, therefore, occurs at a time when the ileum lacks the protection of neutralizing food and secretions. Thus favorable conditions are found for ulceration.

Acute perforation, comparable to that which occurs in the stomach, is found in

TABLE III
HETEROTOPIC

Case No.	Age	Symptoms	Signs	Operative Findings	Pathologic Findings	Rx
			Gastric Mucosa with Ulcer and Hemorrhage			
48	8 mo.	Anemia and 2 episodes of melena	Pallor	Meckel's diverticulum 1.7 by 1.6 cm.	Ulcer on proximal edge of gastric mucosa lining diverticulum	Meckel's diverticulum resected
			Gastric Mucosa with Ulcer and Perforation			
2	6	Right lower quadrant pain with vomiting, 1 day	Spasm and tenderness in right lower quadrant; tender on right on rectal examination	Free fluid perforation of Meckel's diverticulum	Meckel's diverticulum lined with gastric mucosa; perforated ulcer near base	Meckel's diverticulum resected
31	17	Crampy right lower quadrant pain spreading through abdomen 2 days, vomiting	Generalized spasm and tenderness; rectal tenderness	Free fluid; perforated ulcer of Meckel's; stalk ran to umbilicus	Meckel's diverticulum lined with gastric mucosa; perforated ulcer near base	Meckel's diverticulum resected
46	9	Generalized abdominal pain with vomiting 12 hr.	Generalized spasm and tenderness	Perforation at tip of Meckel's diverticulum; free fluid in abdomen	Meckel's diverticulum lined with gastric mucosa; ulcer at tip	Meckel's diverticulum resected
49	24	Upper abdominal cramps 8 days; right lower quadrant pain, 20 hr.; no nausea, vomiting	Generalized spasm and right lower quadrant tenderness	Free fluid in abdomen; perforation at base of Meckel's diverticulum	Gastric mucosa present; ulcer perforated at junction prox. and mid. 1/3	Meckel's diverticulum resected
			Gastric Mucosa without Ulceration			
14	10	Periumbilical pain with vomiting 48 hr.	Right lower quadrant spasm and tenderness	Appendix acutely inflamed; Meckle's diverticulum present	Acute appendicitis; Meckel's diverticulum contained gastric mucosa in diverticulum of its own	Meckle's diverticulum resected
17	21	Hirsutism	Hirsutism	Meckel's diverticulum found durexpl. laporotomy for casue of hirsutism	Gastric mucosa found in tip of Meckel's diverticulum	Meckel's diverticulum resected
			Pancreas			
6	15	Periumbilical pain with nausea and vomiting	Right lower quadrant spasm and tenderness	Ileo-ileal intussusception with Meckel's diverticulum leading	Venous Infarction; pancreatic tissue	Loop with Meckel's diverticulum resected
23	5 mo.	Vomiting, 24 hr.	Melena, visible Peristalsis	Ileocolic intussusception with Meckel's diverticulum leading	Venous infarction; pancreatic tissue	Meckel's diverticulum resected
			Colon			
32	12	Right lower quadrant pain and nausea, 12 hr.	Negative	Meckel's diverticulum 15 inches from ileocecal valve	Meckel's diverticulum lined with colonic mucosa	Meckel's diverticulum resected

Meckel's diverticulum usually in the growing child under eighteen. Perforation in the older age group is rare.

Gastric mucosa was present in seven of our cases and possibly in an eighth. (Table III.) In Cases 14 and 17 there was no associated ulceration; in both of these cases the diverticulum had given no symptoms and was an incidental finding. One patient, Case 48, had ulceration with bleeding and no perforation. This patient was an infant who was studied because of a severe blood-loss type of anemia. The condition was suspected and proved at operation. Four cases and possibly a fifth had gastric mucosa and peptic ulceration with perforation. These Cases are 2, 31, 46, 49 and 50. The last case cannot be included statistically, for the poor condition of the patient precluded removal of the diverticulum. A perforated ulcer was present, but the type of mucosa lining the diverticulum will not be known until a second operation is performed for the removal of the diverticulum. In none of the cases of perforation was there any clue as to the origin of the perforation preoperatively. The patients presented themselves with signs of perforation of a hollow viscus but with no signs which would localize the site of the perforation.

Nodules of pancreatic tissue were present in Meckel's diverticulum in our Cases 6 and 23. In both of these cases intussusception had occurred, with an invaginated diverticulum as the leading point. A casual relationship is suggested but cannot be proved.

Colon mucosa was present in the diverticulum in Case 32. In this instance the finding of Meckel's diverticulum was incidental to other conditions and had no relation to the patient's illness.

DIAGNOSIS

The diagnosis is rarely made preoperatively with any degree of certainty. Symptoms are as varied as the complications and are, in fact, the symptoms of the complications. Patients in whom obstruction is the manifestation cannot be differentiated from those with obstruction due to other causes, nor can patients in whom perforation occurs be separated symptomatically from those with perforation of other hollow viscera. An exception might be made in the latter instance. Perforation of a Meckel's diverticulum has usually a less abrupt onset than perforation of a gastric or duodenal ulceration and is less likely to show the presence of air beneath the diaphragm. However, it cannot be distinguished from appendiceal perforation unless the appendix has been removed previously. In children peptic ulceration of Meckel's diverticulum is an important cause of intestinal bleeding, and such a diagnosis should be entertained in every case.

X-ray has little to offer diagnostically. According to Pfahler,[13] the diagnosis was not made by x-ray prior to 1934. However, since the advent of serial studies of the small bowel, the roentgenologists have on rare occasions been able to show the presence of residual barium in Meckel's diverticulum.

The cases in this series were reviewed with the intention of discovering any symptoms and signs which would lead one to suspect a diagnosis of Meckel's diverticulum. No specific diagnostic criteria were found.

TREATMENT

The treatment of choice is resection; an attempt should be made to take as wide a base as possible in an effort to remove all heterotopic tissue. This form of treatment was carried out in thirty-eight of our cases. In four cases the diverticulum was invaginated by simple purse-string sutures. Two criticisms of this procedure may be made: First, it creates a polypoid mass which may be the leading point of an intussusception although no such cases have been reported; second, it may leave heterotopic tissue. In eight of our cases the diverticulum was not removed. In four of these the process was extremely small; the serious condition of the remaining four

patients precluded any more manipulation than was absolutely necessary.

CONCLUSION

It has been the authors' experience that the incidence of Meckel's diverticulum has shown an increase paralleling the diligence with which it is sought. We note that the incidence in our series in which Meckel's diverticulum actually caused the patient's illness is considerably higher than the incidence in similar series. Perhaps the structure must literally cry for attention before we seek it. More careful search at operation may perhaps increase our percentage of "incidental" Meckel's diverticulum but at the same time prevent future illness. The authors were surprised to discover that only one case in their series was an infant with melena as the result of peptic ulceration. This may, perhaps, indicate some laxity in the study of children with recurrent attacks of abdominal pain. We believe that any child with frequent attacks of abdominal pain which have never been so severe as to bring him to operation should be studied for melena. Any patient whose operative findings fail to substantiate the preoperative clinical impressions should be considered as a prospective victum of Meckel's diverticulum.

SUMMARY

Fifty cases of Meckel's diverticulum are presented and analyzed. The literature is consulted for comparison of this series with the series of other authors. The embryologic background is briefly given. The authors' experience in dealing with these patients is summarized and suggestions are made for improvement in diagnosis and treatment.

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PERCUTANEOUS FIXATION OF HIP FRACTURES

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FRACTURES of the hip, either through the major trochanter or the neck of the femur, occur frequently in older people. These fractures result in the highest mortality of any simple fracture of the long bones and even if the patient survives the result is often poor. Treatment of hip fractures is still a major problem.

Older people do not tolerate well prolonged immobilization which frequently causes intractable bedsores, pulmonary infections, thrombophlebitis and other complications and which, even with the best of care, is depressing and unbearable torture for many of them. In spite of these drawbacks treatment by extension or plastercasts is still used in a great majority of all hip fractures.

In the other extreme are treatments for hip fractures which require cutting surgical procedures, such as application of the Smith-Petersen nail, Moore pins, Lorenzo screws, Bohlman screw pins and related metallic or bony screws, pins, nails or brackets. These operations do away with prolonged immobilization but the necessary anesthesia, loss of blood and surgical shock are frequently too much for these older patients even if the procedure is performed quickly and efficiently.

In between the conservative and surgical methods lies application of screws or pins which are applied without cutting through the skin into the bone uniting the fragments. The idea of application of nails, screws, pins or wires through the trochanter into the neck for the purpose of uniting the fragments is an obvious one and not new. Von Langenbeck (1850), Koenig (1875) and Trendlenburg (1878) already described attempts in this direction. Nicolaysen of Oslo used a percutaneous nail in 1879, Schilling used a square percutaneous nail in 1915, Neufeld described a percutaneous pin, Telson and Rauschoff used several percutaneous steel wires and Gaensler applied subcutaneous spikes. Doubtless in selected cases at present a great number of surgeons drill threaded or plain Steinmann pins into hip fractures through the skin.

The reason that the percutaneous use of pins in hip fractures is not accepted more widely lies chiefly in the technical difficulty of removing the excess length of the pin which extends from the bone through the soft tissues to the outside. It is possible, of course, to push back the skin and muscle to a considerable extent and to cut or file the pin off fairly deep and let the skin slip back. It is impossible, however, to cut the pins off short enough. An inch or more will still protrude from the bone, causing pain and preventing the patient from lying on the affected hip. After the muscles atrophy, as is usual in these fractures, the ends of the pins will protrude under the skin quite visibly and cause distress and possibly perforation.

A primitive method to facilitate removal of the proximal part of the pin consists in filing a deep circular groove around the pin approximately 4 inches from the tip and to break the proximal shaft off by bending it sideways after the pin has been properly applied. This is sometimes quite simple. We found, however, that the pin is loosened too much through bending when the bones are frail. Furthermore, it is difficult to bend the pin in stout or muscular patients. Lately such "break-off pins" have been developed which carry a bead.

Figure 1 shows an assembled new screw pin* which the author designed for the

* The author wishes to thank Edward Weck and Company, Inc., Brooklyn, N. Y. for their cooperation in manufacturing and supplying these pins.

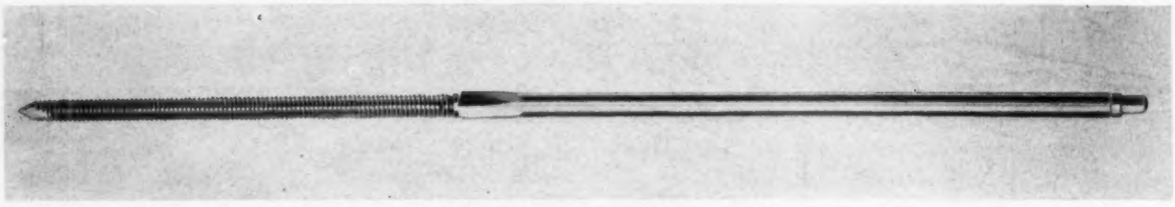

Fig. 1. Pin assembled.

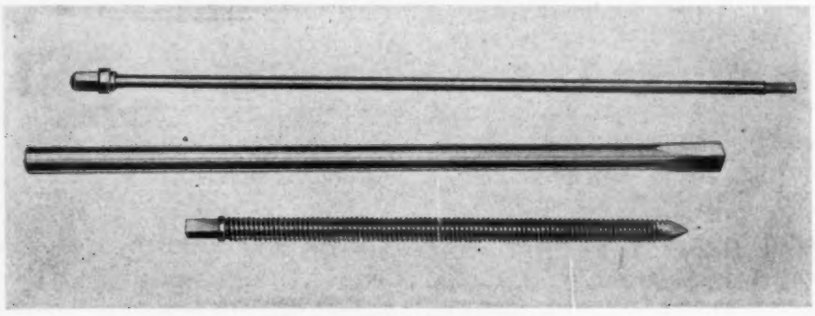

Fig. 2. Pin taken apart.

percutaneous fixation of hip fractures and Figure 2 shows the same pin taken apart. The assembled screw pin is driven into the fracture as deep as necessary. After the position has been found satisfactory the central wire is unscrewed permitting withdrawal of the drive shaft. In this way the screw can be driven into the bone without incision and the shaft can be removed with ease. Figure 3 shows an x-ray of a transtrochanteric fracture immediately after application of the screw and withdrawal of the shaft. The same fracture which is healed four months later is illustrated in Figure 4. The screws are still in the bone and are healed well into their position. The patient, a man of seventy-six years of age, had no external fixation of any kind and walked on crutches six days after the accident. A total of twelve patients with hip fractures were treated at our hospital during the last eighteen months by these percutaneous screws with detachable driveshafts with persistently good results.

It is best to have at least three sterile completely assembled detachable screw-pins of suitable lengths. In addition there should be a liberal supply of $\frac{3}{16}$ of an inch Steinmann, $\frac{3}{32}$ of an inch Granberry pins and some Kirchner wires to be inserted first for the purpose of orientation. Sterile forceps should be ready for insertion of the pins or wire into the drill. Any surgical hand drill or a standard electric drill can be used; only the chuck need be sterilized.

Since the whole procedure is related more to a hypodermic puncture than to an operation, no elaborate aseptic precautions are necessary except, of course, painstaking protection of the asepsis of the screws and pins to be inserted. A pointed knife for making skin punctures and a few skin clips to close them complete the armanentarium. In stout patients it may be necessary to insert several pins or wires before hitting the proper plane. Repeated x-ray controls are necessary. We have, therefore, inserted these pins in the x-ray room either on the Bucky table for stout patients or without Bucky for those who are not as stout. In such instances it is practical to place under the injured hip a box easily made from two plywood sheets held apart on three sides by wooden strips and just large enough to accomodate the 8 by 10 cassette. In this way the film will always be in the same place and the patient does not have to be raised to place the cassette under the fracture side.

Careful reposition with x-ray control is, of course, the first necessary step. The

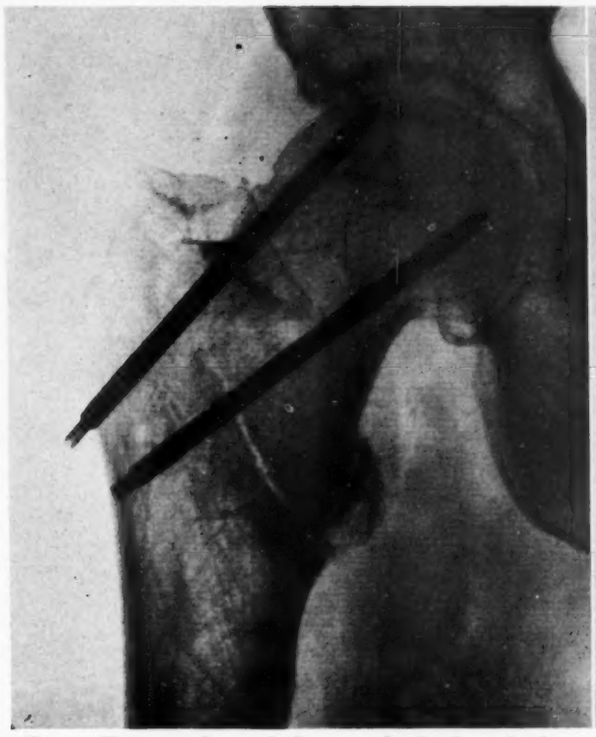

Fig. 3. Transtrochanteric fracture fresh after pinning.

Fig. 4. Same fracture as Figure 3 four months later.

anesthetic should be continued on a light plane; the leg is held in abduction and is rotated inwardly. The skin is carefully prepared and a Kirschner wire is drilled for orientation. If the wire will enter the bone on a steep angle, it will slide off easily and it is simpler to use Steinmann pins for guidance. The first pin may be fully off the intended direction; the second, third or fourth should give enough indication to place safely and accurately the final screws. These are driven assembled into the bone until the resistance by the slightly larger shaft is felt on the bone. After final control x-ray the guide pins and shafts of the screws are removed and the puncture holes are closed by skin clips. No external fixation should be necessary.

The patients can sit up soon after this procedure and they may be permitted to walk on crutches in selected instances. One of our patients, a seventy year old spinster of 105 pounds, was carried into our hospital shortly after she had sustained a complete fracture of the base of the neck of the femur. One hour after admission a reposition was done with ether and two screws were inserted as has just been described. The same afternoon the patient was found walking in the corridor and she was discharged the next day since we could not keep her in bed. A week later she was visited at home and was found doing light chores. She stated at that time that she had been up and around all week and that she was not sure which hip she had broken. She was followed-up regularly and x-ray after four months showed the hip fracture to be well healed and in good position. This case is quite exceptional and shows the utmost contrast as compared with the torture of enforced immobilization in a cast or extension for three or four months.

The treatment of hip fractures by internal fixation and the indications therefor are well established and do not have to be digested here. The purpose of this paper is to describe the method of percutaneous pinning of hip fractures by a relatively minor, non-shocking procedure which re-

quires no cutting, no enforced immobilization and which can be done in a simple way by any general surgeon possessing patience and ordinary skill.

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Bishop et al. studied the healing time of more than 350 bone grafts. Unsuccessful results occurred in about 10 per cent of the cases. Of the remainder the healing time varied. It averaged about twenty weeks for the dual type grafts, twenty-seven weeks for the average inlay graft and twenty weeks for the average onlay graft. Plates were often used in immobilizing these grafts thus permitting earlier ambulation. Healing time was the same, however, whether or not the plates were used. The authors prepared a bed for grafts by removing the outer two-thirds of the bony cortex. They resorted to this technic in the treatment of many cases of non-union of fractures of the long bones of the extremities, with good results. (*Richard A. Leonardo*, M.D.)

TRANSITORY HYPOPROTEINEMIA IN ACUTE SEVERE FRACTURES*

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CURRENT literature abounds in physiochemical studies relative to clinical surgery. Few authors, however, have concerned themselves with the protein alterations in acute severe fractures. In view of this dearth of knowledge our present efforts concerning the protein disturbance in patients with severe fractures appears significantly valuable.

Previous investigations by other authors focused attention on the possibility of protein depletion in trauma and fractures.[1,2,3] Cuthbertson found that nitrogen losses in the urine after fractures reached 25 Gm. daily. Howes and McKeown studied the healing of fractures in rats. They attributed delayed healing to hypoproteinemia. Bone fragility was also tested. They stated that the bones of rats receiving a low protein diet broke after less force was applied to them than did the bones of rats receiving a high protein diet.

Coincident with the early effects of acute trauma, a large output of nitrogen may occur in the urine. Taylor and associates maintained that a large part of this output may be in the form of "undetermined" non-protein nitrogen. They did not believe that this source of nitrogen loss, and hence protein depletion, should be attributed to substances such as urea which are usually found in the urine.

Our own studies (Table 1) have led us to believe in a definite physiologic pattern which occurs following an acute severe fracture. The occurrence of a severe fracture with marked soft tissue damage or destruction is associated with a local loss of

TABLE 1

	Identifying Letters	Age and Sex	Injury	Duration of Injury (Hours)	Shock	Initial Protein Level	Uric Acid Level	Protein Level at Twelfth-hour Interval
1	E. S.	55, M	Compound lower ⅓ left tibia-fibula	4	Absent	4.0	2	6.1
2	A. N.	50, M	Compound supracondylar left femur	3	Absent	4.5	1.5	6.3
3	R. G.	49, M	Lower ½ right tibia and fibula	3½	Absent	4.2	1.5	6.5
4	F. C.	28, M	Middle ⅓ left tibia and fibula	4	Absent	4.0	2.5	6.0
5	A. B.	35, M	Plateau and upper ⅓ right tibia	2½	Absent	5.0	1.0	7.0
6	J. T.	54, M	Comminuted right tibia and fibula	3	Absent	4.0	2	6.8
7	M. C.	32, M	Comminuted lower ⅓ femur	4½	Absent	4.5	1.5	6.5
8	T. A.	60, M	Compound middle ⅓ left tibia and fibula	5	Absent	4.0	2.3	6.0
9	L. Mc.	29, M	Compound lower ⅓ right tibia and fibula	4	Absent	4.5	1.5	6.2
10	A. T.	50, M	Comminuted upper ⅓ right tibia and fibula	6	Absent	4.0	2.0	6.5
11	T. M.	25, M	Compound lower ⅓ left tibia and fibula	5	Absent	4.0	1.0	6.0
12	D. M.	32, M	Compound subcondylar right femur	4	Absent	4.5	1.5	6.2

* From Fracture Service of Dr. John F. Raycroft, Department of Surgery, Kings County Hospital, Brooklyn, N. Y.

Figs. 1 to 4. Illustrative cases of the type of fractures studied in this survey.

protein. In addition there is protein loss in the urine as proven by others who have studied urinary nitrogen loss following trauma.[3] Both sources of protein loss occur prior to the onset of shock. The presence of hypoproteinemia facilitates and predisposes to the onset of shock.[4] For this reason all our cases have been studied prior to the onset of clinical shock.

This hypoproteinemia is believed to be transitory and of insufficient severity to stimulate endogenous protein metabolism

which is the body's mechanism of replenishing a protein deficit. In previous publications attention was brought to the so-called systemic response on the part of the body in order to correct marked hypoproteinemia.[5,6,7]

Hypoproteinemia of this type may be associated with a hyperuricemia which results from accelerated endogenous protein metabolism. In acute severe fractures the protein loss is not severe enough to stimulate this accelerated endogenous physiology. Hence it has been termed "transitory hypoproteinemia."

In our survey twelve patients (Table 1) with acute severe fractures were studied. (Figs. 1 to 5.) All of our patients were adult men, apparently in good health before the onset of their injury. Initial protein studies were made as soon as the patient was admitted to the hospital. If the patient was in shock or displayed any evidence of incipient shock, he was not a candidate for this study. Men with multiple injuries or marked blood loss were likewise not studied. All cases presented had solitary acute severe fractures. In view of the absence of shock, none of the patients studied received parenteral fluid or plasma before or after the fracture was treated. Hematocrit determinations were within normal limits before and after correction of the injury.

Laboratory determinations demonstrated a lowering of the total protein level. Uric acid studies were made at the same time that a protein level was determined. It was found that uric acid remained at a normal value. This indicated that there was no tendency to excessive endogenous protein metabolism. Multiple protein determinations made following injury at hourly intervals indicated that the protein level will return to normal in about twelve hours. During this twelve-hour period the fracture was treated and the patient ate a full meal. If the patient ate before the twelve-hour period, the protein level returned more quickly to normal. Hemoconcentration could not explain the uniform return of the protein to normal levels because of the normal hematocrit valves before and after the fractures were treated. These findings further substantiate the contention that this type of hypoproteinemia is transitory and mild in type. It is not sufficiently severe to demand rectification on the part of the body by stimulating endogenous protein metabolism.

Fig. 5 Illustrative case of the type of fractures studied in this survey.

Not one of the twelve patients studied went into shock following the return of the protein level to normal. This finding is consistent with the knowledge that hypoproteinemia increases the susceptibility to shock.[4] Our current study argues for the old dictum of correcting fractures as soon as possible, not only because a more satisfactory anatomic result may be obtained but also because, in view of the facts presented, shock can be prevented even in severe fractures. This present study corroborates the work of others who demonstrated protein loss following acute trauma.[1,3] In addition our observations emphasize the transitory nature of the hypoproteinemia and the facility with which it can be corrected.

SUMMARY AND CONCLUSIONS

1. Twelve male patients with acute severe fractures were studied from a physiochemical viewpoint.

2. Shock was absent in all instances; hematocrit valves were within normal limits before and after the fracture was treated.

3. All displayed hypoproteinemia without hyperuricemia.

4. The hypoproteinemia is attributed to local protein loss due to tissue destruction and excessive urinary nitrogen excretion.

5. The transitory nature of this hypoproteinemic state is emphasized since a normal protein level was obtained within twelve hours following the correction of the fracture.

6. Reduction of the fracture plus an adequate intake of exogenous protein are the rehabilitating factors in correcting hypoproteinemia.

7. This return to positive nitrogen balance is a cardinal factor in the prevention of shock following severe fractures.

8. In addition to anatomic reasons, the early treatment of fractures is advocated in order to re-establish the physiochemical harmony which may cause severe fracture complications.

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Case Reports

REFLEX ANURIA

ITS TREATMENT BY PROCAINE SYMPATHETIC BLOCK

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THIS work concerns a case of complete anuria and uremia believed to be due to spasm of the afferent arterioles of the glomeruli. Procaine anesthetic block of the sympathetic nerves to both kidneys was followed on two separate occasions by a sudden and dramatic outpouring of urine by both kidneys. It is reasonable to assume that when a large number of glomeruli in a kidney fail to function from any cause, the resulting metabolites may set up afferent impulses which are relayed to the sympathetic nervous system in such a way as to produce spasm in the afferent arterioles of the remaining glomeruli. Whether or not the anuria is due entirely or partially to spasm of the arterioles one would expect relief from a procaine block of the sympathetic nerves to the kidney.

The sympathetic nerve supply to the kidneys has been reported by White and Smithwick[6] as passing through the eleventh and twelfth thoracic sympathetic ganglia. According to Wright,[7] the sympathetic fibers come through all the dorsal ganglia from the sixth to the twelfth, inclusive. Peters[4] believes that vasoconstriction plays an important rôle in reflex anuria. Among the many causes that he has considered for this condition are: obstructing calculus of one ureter, trauma to the renal areas, perinephritic abscess and peritonitis. As early as 1922, an effort was made to combat renal vasoconstriction in reflex anuria by splanchnic procaine block. Peters credits Neuwirt with being the first to successfully treat such anuria by this means. Rubritius[5] reported success with this measure in seven of eight cases. As an alternate procedure to splanchnic block a few writers have advocated spinal anesthesia. Peters reviews reports of Hayes and Paramore, and Cubitt[1] who have reported successful treatment by means of spinal anesthesia.

Decapsulation of the kidney has been used with varying results in cases of nephritis, non-obstructive anuria, post-eclamptic anuria and similar conditions in which the method was considered either a life saving measure or of definite value. Nichol[3] states that decapsulation breaks up the sympathetic connections between the cortex and the capsule and thus in reality is a partial sympathectomy.

TECHNIC

The method used to interrupt the sympathetic nerve supply to the kidneys consists of blocking the two lower thoracic and the two upper lumbar sympathetic ganglia with procaine according to the second method of Labat.[2] The patient is placed either lying flat on the abdomen or turned on the side opposite the one to be injected. Four wheels are made in the skin with procaine 3 cm. lateral to the spine. Two of these are placed in the dorsal region opposite the tenth and eleventh thoracic spines and two are placed in the lumbar region opposite the upper edge of the first and second lumbar spines. The needles used are size 8 to 10 cm. Each needle is inserted vertical to the skin to a depth of 3 to 4 cm. Here the transverse process is contacted. The needle is then passed above

the transverse process and slightly medial until it comes in contact with the vertebral body approximately 3 cm. further forward. A marker in the form of a small piece of rubber should be placed on the needle. When its tip contacts the transverse process, this marker should be placed on the needle 3 cm. from the skin. The tip of the needle is pushed forward toward the body of the vertebra for a distance of 2.5 cm. to 3.5 cm. in front of the transverse process. The needle should not be attached to the syringe when it is being inserted. After it is put in place it should be examined very carefully to see that no spinal fluid or blood escapes. A syringe is then attached and aspirated. The needle should then be rotated 180 degrees and aspirated again. If no blood, spinal fluid or air is obtained, injection may be started.

Five cc. of 1½ per cent solution of procaine is injected into each needle. One should then wait for five minutes, at the end of which a further 5 cc. is injected. The legs are then tested for evidence of anesthesia. If any procaine enters the spinal canal through an anomalous extension of dura, evidence of this will be obtained by signs of anesthesia in the legs. If none is found, one may then inject a further 10 cc. in each needle. The same procedure is repeated on the opposite side.

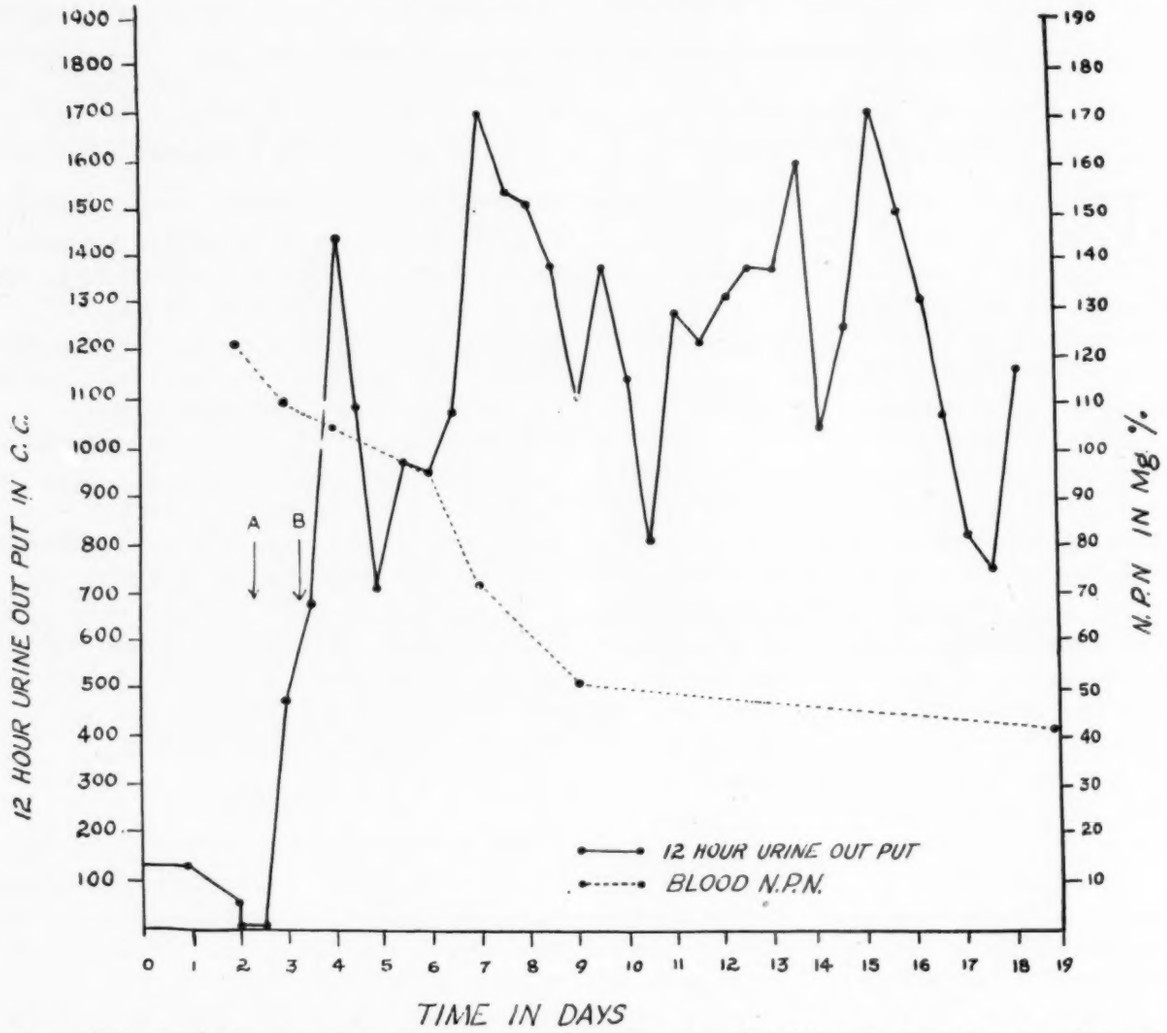

Fig. 1. This shows the urine output following procaine infiltration of the thoracic eleventh and twelfth and lumbar first and second sympathetic ganglia. The procedure was performed at A and again twenty-four hours later at B.

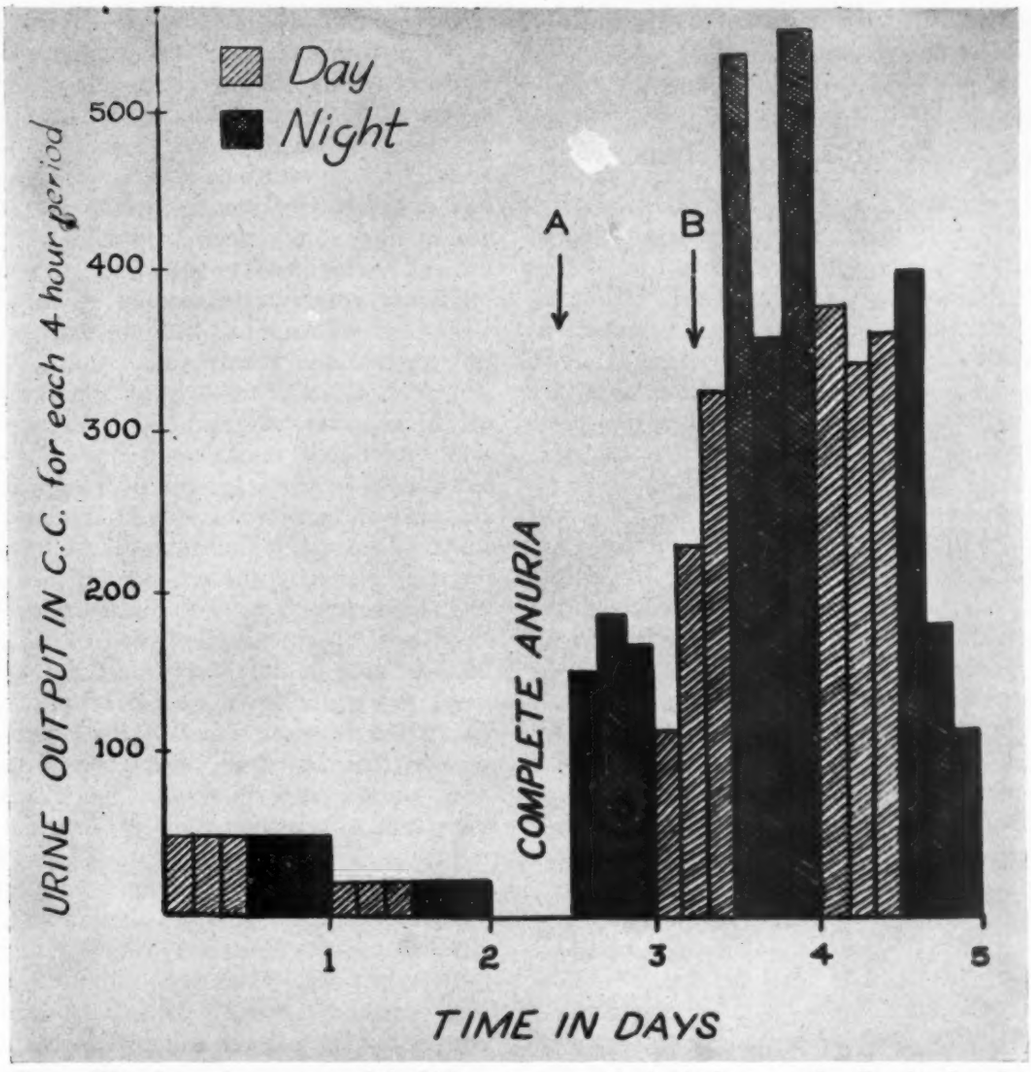

Fig. 2. This shows the response of the kidneys to sympathetic block at A and B. By showing the urine output in four-hour periods one can see more readily the response after each injection.

One must use care in order to prevent puncturing the pleura and so producing pneumothorax. This accident is prevented by keeping the point of the needle close to the body of the vertebra. The injection is done on both sides. The large quantity of procaine used enables one to infiltrate the splanchnic nerves as they pass downward in the lower part of the thorax and upper abdominal region. Also the nerve fibres around the renal artery are infiltrated.

RESULTS

The results were very gratifying. This patient had not passed very much urine for fifty-six hours. For the twelve hours preceding the first treatment he had passed no urine. (Figs. 1 and 2.) Within an hour of the first sympathetic block, he was passing urine at the rate of 28 drops per minute. This rate continued for twelve hours during which time he passed a total of 480 cc. He continued to excrete urine for the next twelve hours at a reduced rate, finally reaching a level of only 7 drops per minute. The sympathetic block was then repeated with even better results. The urine flow promptly increased to 40 drops a minute. This continued for twelve hours during which time he passed a total of 1,445 cc. of urine. The urine flow continued at this rate for another twelve hours and

then settled down to a normal rate of flow. The blood non-protein nitrogen which had risen to 120 slowly decreased to a level of 50 mg. per cent. At the time of this report the patient's condition is excellent.

CASE REPORT

A white male, age nineteen, was admitted to Regional Hospital, Camp Joseph T. Robinson, Ark., on January 16, 1945. His chief complaints were malise, chills and fever. He stated that he had had a cold since induction two monthly previously. He went on sick call the day prior to admission and was assigned to quarters because of malaise and fever. That night he felt increasingly worse and developed chills and fever. The following day he was admitted to the hospital. On admission he had considerable malaise and a mild cough which was slightly productive of yellowish sputum. There were no other symptoms except for moderate headache.

Physical examination on admission revealed a well developed and well nourished individual not appearing acutely ill. The temperature was 100°F., the pulse 88 and the respiration 20. The blood pressure was 118/80. The pharynx was congested but no membranes were seen. The tonsils were injected and moderately enlarged. Moderate bilateral cervical adenopathy was present. Examination of the chest revealed no abnormality of the heart or lungs. The abdomen was normal except for a healed incisional scar of the left upper quadrant. The remainder of the physical examination was essentially normal. The family history was non-contributory. His past personal history revealed that he had measles and mumps in childhood. He had recurrent attacks of acute tonsillitis four or five times in the past three years. The first attack was the most severe during which time he was in bed one week. No history of scarlatina, pneumonia, influenza, rheumatic fever, nephritis or other illnesses.

On admission to the hospital the patient was considered to have moderate nasopharyngitis and acute bronchitis. He was treated symptomatically with bed rest for two days after which he felt quite good and was permitted bathroom privileges. The temperature ranged from 98.6 to 99.8°F, for five days. On January 21st, the temperature rose to 103°F., pulse to 100 and respirations to 26, but they returned to normal limits by January 22nd. On January 24th, eight days after admission, his temperature again rose to 101.2°F. Examination of the lungs revealed diminished breath sounds and scattered fine crepitant râles at the right base posteriorly. X-ray of the chest on this date showed a pneumonic process in the right lower lobe which was considered to be an atypical pneumonia. The general appearance of the patient remained good. The temperature gradually returned to normal. Urinalysis on admission was normal, but on January 26th the microscopic examination showed 4 to 6 white blood cells and 8 to 10 red blood cells per high power field. On January 27th, urinalysis was again normal except for 2 or 3 red blood cells per high power field. On January 29th it was normal except for an occasional white blood cell. The total blood leukocyte count on January 26th was 12,500.

On February 3rd the patient had been afebrile for eight days. X-ray on this date showed considerable resolution of the pneumonic process in the right lower lobe. Because of a painful tooth of two days' duration, he was referred to the dental clinic where extraction of L 4 was performed. On the evening of February 6th, he complained of pain in the right side of his chest. The temperature was 102°F., pulse rate 120 and respirations 40.

On February 7th he was placed in an oxygen tent because of slight cyanosis and increased respiration rate. Physical examination at that time revealed evidence of a pathologic disorder at the right lower lung base with dullness on percussion, diminished breath sounds and numerous subcrepitant rales. Penicillin therapy was begun. On this date it was noted that no urine had been passed for the preceding twenty-four hours in spite of a total fluid intake of 4,320 cc. For this reason he was catheterized and only 270 cc. of dark red urine was obtained. An indwelling catheter was left in the bladder in order to follow more closely the kidney function. In the next twenty-four hours the output of urine was only 150 cc. in spite of an intake of 2,680 cc. Blood non-protein nitrogen was 120 mg. per cent. During the next twelve hours the patient put forth no urine at all despite adequate hydration. On February 8th physical examination indicated an extensive pleural effusion of the right side of the chest. This finding was corroborated by portable x-ray. Thoracentesis was performed and 1,020 cc. of turbid, straw-colored fluid was obtained

from the right pleural space. Penicillin, 40,000 units, was injected into the right pleural space. Smear and culture of the pleural effusion was later reported negative. Laboratory tests on this date revealed the following: Urinalysis: specific gravity, 1.023; acid reaction; albumin, 3+; sugar, negative; microscopic, innumerable red cells. The blood counts were: red blood cells, 4,100,000; white blood cells, 24,400. Differential: neutrophiles, 87 per cent; lymphocytes, 13 per cent, Blood non-protein nitrogen, 126 mg. per cent. Alkalinization of the urine by sodium citrate was instituted. Penicillin was administered intramuscularly, 20,000 units every two hours. Clinically the patient appeared acutely ill. There was some hiccoughing which was considered secondary to nitrogenous retention. Blood chloride level was 440 mg. per cent.

In the evening of February 8th paravertebral sympathetic block was performed. The lower two thoracic and upper two lumbar ganglia were infiltrated with procaine 1½ per cent. Twenty cc. were injected at each site. Following the sympathetic block, urine output increased dramatically within two hours. During that night there was a total output of 480 cc. On the afternoon of February 9th, the urinary output again decreased to 7 drops per minute from the indwelling catheters. A sympathetic block was performed as before with even better results. Urine output suddenly increased to 40 drops per minute. During the next twelve hours there was a total output of 1,445 cc. The urine output continued at this rapid rate for another twelve hours and then decreased to a normal rate as indicated in the graphs. (Figs. 1 and 2). The next day the hiccoughing disappeared. Thereafter the patient had a stormy course but with gradual improvement. Intravenous fluids were used to supplement oral intake so that dehydration was never present. Thoracentesis was performed on February 10th when 1,000 cc. of turbid, straw-colored fluid was removed. On the same day, because of nausea, vomiting and distention, Wangensteen suction was instituted.

On February 26th, because of secondary anemia with red blood cell count of 2,850,000 and hemoglobin of 53 per cent, a transfusion of 500 cc. of citrated blood properly typed for hemologous grouping and Rh factor was administered, but was discontinued after 300 cc. had been administered because of slight chills and nausea.

Thereafter, there was slow but progressive improvement in the general condition of the patient with the exception of continued albuminuria 1+ to 3+ with numerous red blood cells which gradually disappeared. The pathologic condition in the right lung rapidly disappeared.

COMMENTS

The large quantity of procaine injected to accomplish the sympathetic block in this case was used for two reasons. First, because smaller quantities had been unsuccessful in relieving a case of reflex anuria previously treated in this hospital. Secondly, because the innervation of the kidneys is believed to come from the lower six thoracic sympathetic ganglia, it was considered necessary to block the splanchnic nerves as they pass downward in the chest and abdominal cavities. The question arises as to whether or not this patient would have recovered without a sympathetic block. It is very probable that he would not have recovered since he was rapidly going down hill in a condition of uremia. The results were too sudden and dramatic to be an accident. By consulting the graph in Figure 2 one will note that the night output following sympathetic block on both occasions far exceeded that of the day. This is the reverse of what would naturally occur if the return of kidney function was spontaneous and not the result of the block. As it was the sympathetic block was performed in the afternoon and so the greatest output was immediately following.

This procedure will probably not benefit cases of anuria in which spasm of the arterioles is not a factor. Because the procedure is so safe and easy to apply in the very ill, it should be given a trial even when possibility of benefit is remote before submitting the patient to the more major procedure of decapsulation. Particularly is this so when the condition is of a critical nature.

If the first block does not produce benefit, it should be tried for two more occasions at twelve to twenty-four-hour intervals since we have found that in treating conditions due to a disturbance in the sympathetic nervous system each successive treatment up to the third is more beneficial than the one before. This patient was well hydrated at the beginning of the oliguria since he had received 4,320 cc. of fluid in the preceding twenty-four hours. After anuria became established the daily fluid intake was cut to 2,680 cc. in order to avoid overhydration. The increase in renal output was accomplished without increase in intake; however, after renal function was restored by novocain sympathetic block the fluid intake was increased.

SUMMARY

1. Reflex anuria due to a spasm of the arterioles of the glomeruli is believed to occur with retroperitoneal hemorrhage, injury to one or both kidneys or in cases in which organic disease within the kidney actually exists.

2. A case is reported in which reflex anuria was relieved by blocking the sympathetic innervation to the kidneys with large quantities of procaine.

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POLYPOSIS OF THE STOMACH TREATED BY TOTAL GASTRECTOMY

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GASTRIC polyposis was first described as a disease entity by Cruveilhier[4] in 1833. Quain[7] in 1857 presented a case to the pathological society of London of a young girl who vomited a gastric polyp the size of a chestnut; this is believed to be the first such diagnosis made during the life of a patient. Chosrojeff[3] in 1912 is credited with having made the first clinical diagnosis from gastric lavage which contained particles of polypi. According to Balfour,[1] Carman in 1919 was the first to demonstrate the value of x-ray in the diagnosis of this condition, and Schindler[8] in 1922 is credited with the first gastroscopic diagnosis. Since that time with improving x-ray and gastroscopic technics, gastric polyposis has received increasing attention in the literature. Brunn and Pearl,[2] in a comprehensive review in 1926, were able to find only eighty-four proven cases of gastric polyposis to that date but were able to add forty-one more cases by 1943.

Isolated polyps of the stomach are not at all unusual; in fact, various authors estimate that about 1 per cent of all tumors of the stomach are of this type. The term polyposis has been arbitrarily applied to cases in which three or more polyps are present simultaneously in the stomach and although many classifications have been advocated there would appear to be two general types of polyps from a gross and microscopic point of view. A detailed description of the differences in the pathologic picture of these two types can be found in the work of Menetrier.[6] However, the salient features of each variety as described by him would seem to be as follows: The first type, "polyadenomes polypeaux," which consists of polyps that have a slender stalk and head with normal gastric mucosa present between the polyps. Microscopically, this group is characterized by the presence of submucosa and muscularis extending out into the stalk. Cyst formation is common. The second type, "polyadenomes en nappe," is a slightly elevated growth having a sessile base resting on the submucosa. There is no differentiation into a head and stalk as in the first type and no normal gastric mucosa is present between the polyps. In fact, much of the gastric mucosa shows evidence of chronic gastritis either of the hypertrophic or atrophic type.

Polyposis of the stomach has been variously described as having been found in individuals ranging in age from twenty-three to eighty. Epigastric discomfort is usually the earliest complaint although at times vomiting, hematemesis, blood in the stools and diarrhea may be associated. The incidence of hypochlorhydria and even achlorhydria in polyposis of either type is extremely high, and it might be worth noting that polyposis of the stomach is present in many patients with pernicious anemia in whom chronic gastritis and achlorhydris are commonly found. Roentgenologically, the diagnosis may be extremely difficult and, in fact, Brunn and Pearl state that in their most recent series of forty-one cases only seventeen were diagnosed by x-ray. The most positive method of diagnosis is by gastroscopic visualization of the lesions.

In the 129 cases reported to 1943 three methods of treatment were described: (1) Gastrotomy with local excision of the polyps. (2) Subtotal gastrectomy consisting of resection of the polyp-bearing portion of the stomach if the polyps were

limited to a relatively small area. (3) In cases in which polyposis was present throughout the entire stomach subtotal gastrectomy was done, supplemented by local excision or fulgeration of the polyps remaining in the cardiac end of the stomach.

It would appear that in cases in which extensive polyposis involves the greater portion of the stomach, particularly if associated with marked gastritis or when most of the polyps are situated in the cardiac end of the stomach, the aforementioned methods of treatment would be highly unsatisfactory. It would appear further that nothing short of total gastrectomy could offer such an individual a permanent cure, particularly since these polyps are regarded as precancerous lesions with actual malignancy being present in approximately 20 per cent of autopsy specimens.

We would like to present a case in which the polyps were scattered throughout the entire stomach, but were especially numerous in the cardiac portion, and in which moderate gastritis of the entire gastric mucosa existed. This patient was treated by total gastrectomy and although this procedure is being performed with increasing frequency for carcinoma of the stomach we have been unable to find any other case report in which gastrectomy has been performed for polyposis.

CASE REPORT

J. M., a white man aged sixty, was admitted to the Bryn Mawr Hospital October 14, 1945. This man had first consulted a physician in May, 1945, with the complaint that for several months he had noticed increasing fullness after meals, anorexia, belching and loss of energy and weight. His past medical history was irrelevant except for two factors: In 1916 he had been operated upon for acute appendicitis with perforation, following which he developed thrombophlebitis of the left leg with pulmonary embolus, and in 1937 an episode of jaundice occurred for which he was treated medically with no recurrence.

During the first few weeks under his physician's care numerous studies were carried out and were negative, with the following exceptions: An x-ray of the stomach on June 21, 1945, suggested polypoid change of the gastric mucosa; gastric analysis June 22, 1945, showed an achlorhydria and gastroscopic examination by Dr. Frederic Monaghan performed on June 28, 1945 revealed a generalized polyposis of the entire stomach but more especially in the mid- and cardiac portions.

It was believed that the lesion was so extensive that surgical removal of the entire stomach was indicated and a regimen consisting of high vitamin, high protein, and high caloric diet plus use of insulin was instituted in preparation for operation. By the time the patient was admitted to the hospital he had gained over 15 pounds in weight although his original symptoms continued unchanged.

Physical examination on admission revealed a rather pale, gaunt, apathetic man 6 feet tall, weighing 130 pounds. Other than a slight systolic apical murmur examination was essentially negative.

Laboratory examination revealed the following: red blood cells, 5,050,000; hemoglobin, 16.3 gm. or 101 per cent; white blood cells, 9,600; polymorphonuclears, 64 per cent; lymphocytes, 32 per cent; monocytes, 2 per cent; plasma cell, 1; basophiles, 1; urinalysis negative; Wassermann, negative; blood urea nitrogen, 12 mg. per cent; serum protein, 6.4 Gm.; albumin, 4.9 Gm.; globulin, 1.5 Gm.

Operation was performed on November 23, 1945. With continuous spinal novocain anesthesia and through an upper left rectus incision, exploration of the abdomen was carried out and no gross pathologic disorders could be detected. The stomach was of average size and there was some evident thickening of the anterior gastric wall in the cardiac portion. No definite polypi could be palpated through the gastric wall. In order to determine more accurately the extent of involvement of the stomach by polypi gastrotomy was done through which polypi of various sizes were seen to be present from the pylorus to the extreme cardiac end of the stomach, being more numerous at the latter end. Likewise at the cardiac end of the stomach there was evidence of a definite chronic inflammatory process involving the mucosa. It was believed, therefore, that nothing less than total gastrectomy could offer this patient a permanent cure. Therefore, this

Fig. 1. Specimen of entire stomach containing polypi throughout.

procedure was then carried out according to the method of Lahey,[5] the entire stomach being removed, the duodenum closed, the jejunum anastomosed side-to-end with the esophagus and a jejunojejunostomy performed. Ten Gm. of sulfanilamide were introduced into the abdomen and the abdomen was closed without drainage. The patient received approximately 1,000 cc. of plasma throughout the procedure and his condition at conclusion of the operation was good.

Pathologic report by Dr. Max Strumia disclosed the following: "A. *Gross Description:* The specimen consists of an entire stomach and greater omentum with a portion of the gastrohepatic omentum. The stomach was opened along the greater curvature. Approximately twenty-five fairly discrete polyps are seen to rise from the mucosa a short distance from the cardiac orifice. Most of these are on the anterior and posterior wall adjacent to the lesser curvature. The polyps vary in diameter from 0.5 to 1.5 cm. Some are sessile, others are pedunculated.

"B. *Microscopic Description:* The gastric mucosa adjacent to the polyps shows moderate lymphocytic, eosinophilic and plasma cell infiltration. The cares of the polyps rise from and include the submucosa which contains an increased number of dilated, blood filled veins. The mucosal covering of the polyps is not increased in thickness. The glands and gastric pits show marked distortion and frequent cyst formation. In some of the polyps the pattern of the glands is very atypical and the cells are hyperchromatic. This suggests malignant change, but there is no invasion beneath the muscularis mucosa so that a diagnosis of carcinoma is not justified." Note the entire stomach with polypi in the accompanying illustration.

During the first ten postoperative days the patient's temperature ranged from 99 to 101°F., pulse from 100 to 130 and respirations from 20 to 24. He received daily transfusions of plasma and whole blood for three days and subsequently at intervals of from two to three days to maintain a satisfactory blood count and serum protein level. Sulfadiazine and penicillin therapy were instituted on the day following operation and were maintained for approximately two weeks. He received nothing by mouth for three days at which time feedings were begun with 1 dram of tap water at hourly intervals. During the subsequent week his diet was increased to the point at which he was

receiving fourteen-hourly 3-ounce feedings of surgical liquids, gelatin, junkets and custards. Two weeks following operation white meat of chicken and puréed vegetables and fruits were added to his diet.

We have thought, as have many other surgeons, that early ambulation of patients postoperatively is an important measure in preventing thrombophlebitis, phlebothrombosis, pulmonary embolism, etc., particularly when an earlier history of one of these conditions is present. As just mentioned this man had had an episode of intravenous clotting with pulmonary embolism following an appendectomy in 1916, and for this reason he was gotten out of bed daily from his first operative day. In spite of this on his tenth postoperative day some edema of the left ankle and lower leg appeared although with compression bandaging and elevation of the extremity this was of a minimal degree and persisted only a few days.

The patient was discharged from the hospital on December 5, 1945, with the incision healed by primary intention; his rather long period of hospitalization can he explained by inadequate facilities for care at his own home. He was eating the equivalent of a soft diet with fair appetite and with no discomfort and his weight was 108 pounds.

Since December 5, 1945, the patient has continued to be free of symptoms, his weight has increased to 115 pounds, he has returned to his former occupation and his diet is now practically unlimited.

SUMMARY

1. Although only 125 cases of gastric polyposis have been reported prior to 1943, this condition is receiving increasing attention in the literature because of improving roentgenologic technic and use of the gastroscope.

2. It would appear that treatment up to the present time, which consisted of gastrotomy with excision of the polyps, subtotal gastrectomy or subtotal gastrectomy with fulgeration of the remaining polyps, has been inadequate in cases of extensive polyposis.

3. A case of extensive polyposis with associated gastritis of the entire stomach treated by total gastrectomy is reported.

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LYMPHOBLASTIC SARCOMA OF THE SMALL INTESTINE WITH PRIMARY MANIFESTATIONS IN THE SKIN*

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IN the patient whose case we are here reporting a lymphosarcoma developed in the skin of both feet approximately four and one-half months before there were symptoms caused by the primary tumor in the small intestine.

The records show that malignant lymphoid tumors developing in any part of the body may metastasize to the skin, but we have been unable to find detailed reports of cases in which cutaneous involvement became apparent prior to symptoms referable to the primary tumor.

CASE REPORT

A forty-six year old man was admitted to St. Luke's Hospital November 1, 1945, for treatment of circumscribed ulcerated tumors of both feet, of ten weeks' duration. The first symptom had been a mild burning sensation in both lower extremities. Two weeks after onset, red splotches appeared in the skin of the dorsum of both feet and within four weeks the reddened areas became ulcerated. At the time of admission to the hospital, there was extensive involvement of the soft tissues of the left foot by a fungoid tumor. (Fig. 1.) The lesion of the right lower extremity was limited to a single ulcerated tumor, approximately 4 cm. in diameter, in the skin of the dorsum of the foot. The patient had no complaints except those related to the lesions of the feet. He appeared to be in good health and physical examination was essentially non-contributory, except for the above mentioned tumors, a right inguinal hernia and deafness following meningitis. His temperature, blood count and sedimentation rate were within normal limits. Serologic tests for syphilis were negative. Roentgenograms revealed no bone involvement of the lower extremities and the chest was normal.

A biopsy specimen was taken from the tumor of the left foot. Microscopic examination of the biopsied material (Figs. 2 and 3) revealed an edematous area of skin invaded by a small celled neoplasm. The tumor was composed of irregular cells varying in size from 19 $\mu\mu$ to 24 $\mu\mu$ with poorly defined cell borders and pale homogeneous eosinophilic cytoplasm. Large, oval or reniform nuclei occupied an eccentric position within the cells. The nuclear membrane appeared thin and distinct. The nucleoplasm stained lightly, and the contained chromatin formed a fine, powdery network with numerous small points of condensation. A single prominent nucleolus was usually present. Mitotic figures were abundant in all portions of the tissue. Associated with the above cells there were numerous nucleated erythrocytes, eosinophils and occasional mature lymphocytes. Silver impregnation (-1-) showed fairly well developed argentophilic recticular fibers between the cells. In a few instances the

FIG. 1. Metastatic lymphosarcoma of the left foot on admission.

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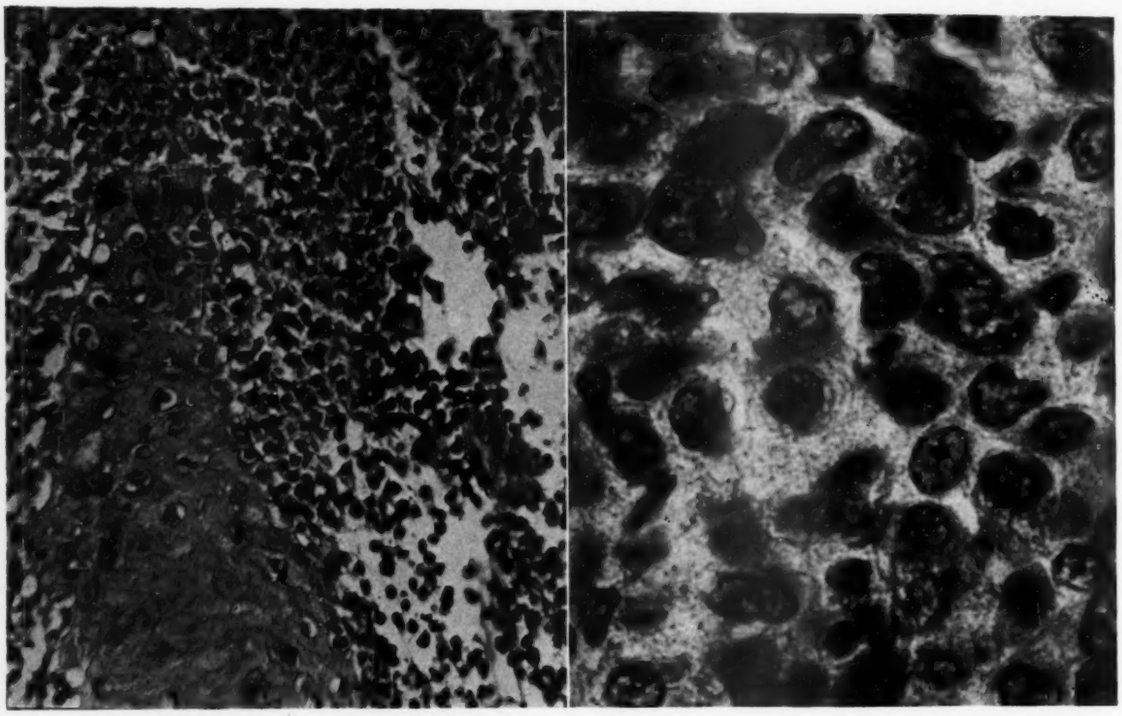

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Fig. 2. (× 1875) Photomicrograph of material removed at biopsy of left foot showing edematous skin infiltrated by lymphosarcoma.
Fig. 3. (× 1250) Photomicrograph showing large lymphoid cells in skin of left foot.

fibers passed through cells resembling those of the tumor but were probably hyperplastic reticulocytes.

A short course of roentgen therapy, totaling 1,500 r. units to the right and 840 r. units to the left foot, caused the tumor of the right to disappear and the tumor of the left to decrease in size. Obviously the cells were very radiosensitive. On December 4, 1945, thirty-four days after admission, the left leg was amputated above the knee because of the hopeless condition of the foot with its advancing and extensive gangrene. The postoperative course was uncomplicated and the patient left the hospital thirteen days after operation.

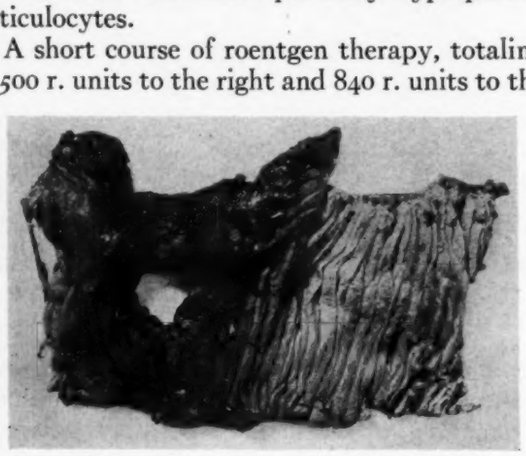

Fig. 4. Primary lymphosarcoma of the intestine with perforation removed at operation.

On January 19, 1946, six weeks after amputation and four and one-half months after the development of the skin lesions, the patient was readmitted to the hospital because of epigastric pain, nausea and vomiting. These symptoms subsided rapidly and roentgenograms of the stomach, duodenum and colon disclosed no abnormalities. Small bowel studies were not made.

On March 8, 1946, the patient experienced a second attack of severe abdominal pain and returned to the hospital in critical condition. There was marked abdominal distention and palpation disclosed diffuse tenderness. Roentgen studies showed distention of the small intestine. Celiotomy revealed a diffuse thickening in the mid-jejunum through which a 4 cm. circular perforation had occurred. (Fig. 4.) The involved segment of bowel was resected and intestinal continuity reestablished by end-to-end anastomosis.

The postoperative course was satisfactory for one week when the patient began to complain of anorexia and abdominal pain. The temperature varied from 99 to 101°F., while the

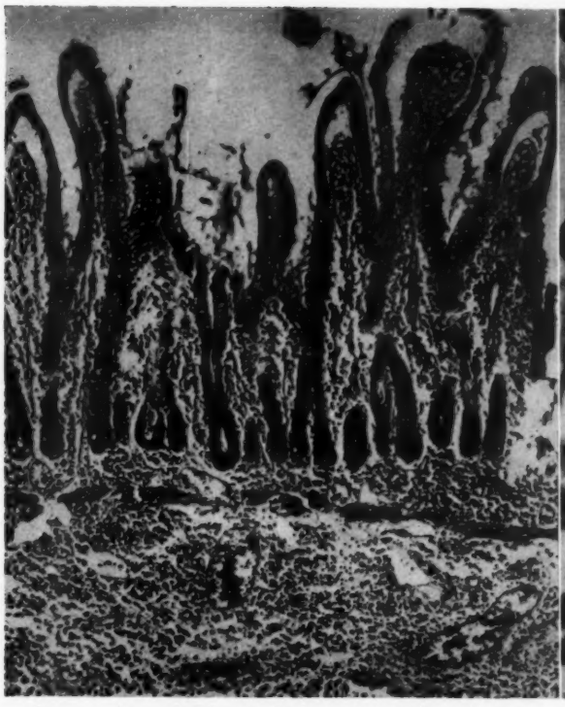

Fig. 5. (× 75) Photomicrograph of the jejunum showing infiltration by lymphosarcoma.

Fig. 6. (× 875) Photomicrograph of a portion of omentum showing lymphoid character of tumor cells.

white blood count showed only a slight elevation with normal distribution of cell types. On rectal examination a firm, non-tender mass could be felt. These symptoms continued for four weeks when a second jejunal perforation occurred. This was treated by another resection. The patient failed rapidly and died two days after operation.

Postmortem examination revealed generalized peritonitis and a third perforation which had occurred in a soft, white, non-constricting tumor mass located 6 cm. above the second anastomosis in the jejunum. The wall of the sigmoid colon was found to be involved by a similar tumor. The stomach, duodenum, ileum, ascending, transverse and descending colon, lung and liver were free of tumor. The mesentery of the jejunum contained numerous soft, white tumor masses and the retroperitoneal lymph nodes appeared to be extensively involved by infiltrating tumor. The spleen weighed 275 Gm. Section of the spleen revealed numerous soft, whitish masses varying in diameter from 1 to 2 cm. Microscopic examination of the jejunal (Figs. 5 and 6) and other abdominal tumors revealed tumor cells which were identical in all respects to those observed in the biopsied specimen from the foot.

COMMENT

The primary tumor in the intestine has the microscopic morphology of a large cell lymphosarcoma of lymphoblastoma, as some prefer. The character of the tumors in the feet is somewhat less clear but the chief element is certainly of lymphocytic type. Certain cells show a close relationship to the argentophil fibers but they are probably connective tissue cells from the neighboring inflamed tissues and do not necessitate altering the diagnosis of lymphosarcoma.

It is generally believed that lymphosarcoma does not develop as a primary cutaneous tumor[2] and the bilateral involvement of the skin of the feet in this case is evidence of a metastatic rather than primary tumor. Although skin lesions developed four and one-half months prior to any intestinal symptoms, it is evident that this tumor originated in the lymphoid tissues of the jejunum. As both of the extremities were involved at practically the same time it is apparent that the metastatic cells

must have entered the circulation above the bifurcation of the aorta.

GENERAL CONSIDERATIONS

The exact classification of the lymphoid group of tumors is not yet settled. Transitional types certainly occur. Gall and Mallory[3] proposed the following classification and nomenclature: stem cell lymphoma, clasmatocytic lymphoma, lymphoblastic lymphoma and lymphocytic lymphoma. These four groups represent a further subdivision of Ewing's older classifications and, as Gall and Mallory point out, they present a fairly satisfactory working basis for the classification of malignant lymphomas.

Malignant Lymphoid Tumors of the Intestine. Malignant lymphoid tumors of the intestine are relatively infrequent. Graves,[4] in 1919, collected 249 cases, and in 1932 Ullmand and Abeshouse[5] added 126 cases, bringing the total of reported cases to 375. The incidence of lymphoid tumors in malignancy of the entire intestinal tract is given by McSwain and Beal[6] in their report from The New York Hospital as 1.9 per cent. Warren and Lulenski[7] report an incidence of 0.9 per cent and Frygole, Kiernan and Docherty[8] state that approximately 1 per cent of all malignant tumors of the gastrointestinal tract, seen at the Mayo Clinic, are lymphoblastomas. Sugarbaker and Craver,[9] in reviewing 196 cases of lymphosarcoma seen at the Memorial Hospital, found that the primary lesions appeared in the gastrointestinal tract in thirty-one, or 16 per cent. Warthin,[10] in reporting 134 cases of lymphosarcoma, states that the primary tumor was in the intestinal tract in twenty-six, or 19 per cent.

Malignant Lymphoid Tumors of the Skin. Ewing[11] believed that lymphosarcoma of the skin occurs only as a metastatic process from other regions and stated that cutaneous lesions might appear early or late in the disease. On the other hand, Warren and Lulenski[7] state that, "Lymphoid tumors of the skin, without evidence of lymph node involvement, have long been recognized and often classified under the broad description of 'Mycosis Fungoides.' These tumors may be the first manifestation and sole evidence of disease for many years. The majority of patients that come to autopsy have similar tumors involving either lymph nodes or other organs." Reported cases of skin metastases from intestinal lymphosarcoma are rare despite the fact that Gates[12] has reported 9 per cent cutaneous involvement in patients dying from malignant lymphoid tumors, and Sugarbaker and Craver[9] found the incidence of skin metastases in their 196 cases of lymphosarcoma to be 5.5 per cent. Neither of these reports indicate how many, if any, of these tumors manifested themselves primarily by cutaneous involvement.

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HODGKIN'S DISEASE OF THE STOMACH

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HODGKIN'S disease limited to the stomach is of sufficient rarity and surgical interest to warrant recording. The following case is of particular value because the writer has had the opportunity of making yearly examinations of the patient for the past thirteen years due to the fact that annual examinations and Kahn tests are required of barbers in this state. The patient is now seventy-one years of age and is still active at his trade. Except for occasional episodes of mild cardiac decompensation, which respond to digitalis and rest, he enjoys good health.

CASE REPORT

Mr. A. O., age fifty-eight, entered Lutheran Hospital on July 26, 1933. The patient stated that he felt well up to three months before admission. At that time he noticed pain in the upper abdomen. This pain would come in attacks; with each attack he would break out into a sweat and the pain would radiate throughout the entire abdomen. There was a weight loss of 10 pounds in these three months. During this time he noticed tar-colored stools on several occasions. His past history and family history were irrelevant.

The patient was well developed, pale and was not acutely ill. The heart rate was 76 per minute with each fifth beat followed by a dropped beat. The blood pressure was 130/85. The abdomen contained a large fixed mass in the epigastrium, extending about 4 inches to each side of the midline and downward nearly to the umbilicus.

Laboratory examination revealed the following: Red blood cells numbered 2,740,000; hemoglobin was 60 per cent; white blood cells numbered 9,000. The differential count was: juveniles, 4 per cent; stabs, 12 per cent; segmented, 65 per cent; lymphocytes, 15 per cent; monocytes, 4 per cent. Red blood cells showed slight poikilo and anisocytosis. Many basophilic stippled cells were seen. The red blood cells had a pale appearance. Urine examination showed albumin, one plus. A few finely granular casts were found.

Gastrointestinal examination by x-ray showed a very large ulcer of the lesser curvature of the stomach, probably a carcinoma.

The laboratory report by Dr. W. Siebert follows: "The frozen section made at the time of the operation shows extensive infiltration of the stomach mucosa, muscularis, serosa and peristomach fat tissue with masses of individual cells which are closely packed and resemble reticulum cells. Scattered between these are typical plasma cells and eosinophiles. There is considerable erosion and ulceration of the stomach mucosa. The lesion apparently arises from the mucosa and is extending through the connective tissue between the muscle bundles. A few definite but somewhat atypical Dorothy Reed cells are seen scattered throughout the tissue. The paraffin section shows typical Hodgkin's granuloma."

The operation was performed on July 27, 1933, by Dr. R. Berg and Dr. C. F. Sherwin. The abdomen was opened through a left rectus incision. The muscle was retracted laterally. Palpation of the stomach region revealed a firm area on the lesser curvature adherent to the liver. No glands or liver metastases were palpable. On dissecting the adherent area of the stomach from the liver, a perforation occurred with the escape of gastric contents. The ulcer was then inspected from within and was found to have a sharply circumscribed margin which extended outward for a distance of about 4 cm. from the perforated area. This entire section was excised in a V-shaped (Fig. 1) manner and the stomach was closed first with a row of sutures from within, then with a Lembert suture from without. Vessels were ligated individually. One drain was placed into the lesser peritoneal cavity and one to the site of the ulcer. The abdomen was closed in layers; the patient was given two transfusions postoperatively. He had a temperature of 102°F. for two days; it then ranged from 99° to 101°F.,

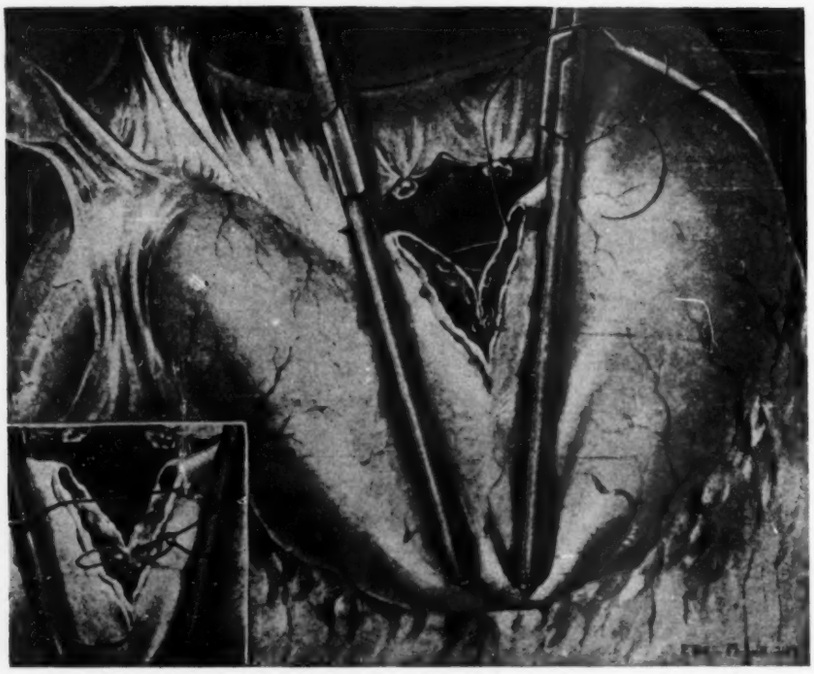

FIG. 1. Excision of an ulcer on or near the lesser curvature of the stomach. The clamps are applied to include the ulcer which is excised after separation of the gastrohepatic omentum. In the small figure the first stitch is shown. (Courtesy of W. B. Saunders Company—Taken from "Abdominal Operations" by Moynihan.)

for ten days. The patient was discharged on the twenty-fourth postoperative day with two granulating areas still in the wound.

COMMENT

Attention was first drawn to gastrointestinal lymphogranulomatosis by the writings of Schlagenhaufer,[1] Terplan,[2] Sternberg[3] and Coronini.[4] Gastrointestinal involvement in Hodgkin's disease may be primary, as in the case here reported, or the involvement may be part of a generalized Hodgkin's disease. The frequency of primary gastric involvement may be judged by the figures of Jackson,[5] who made a careful review of 174 cases of Hodgkin's disease and noted three of this group to be primary in the stomach. This author pointed out the important fact that such lesions may be asymptomatic for some time and finally a fatal rupture of such a lesion may occur. Apparently the lesion in our patient was asymptomatic until it approached the serosa.

The gross appearance of the stomach lesions in the reported cases is very similar, namely, a soft flat infiltrating tumor associated with soft lymph nodes in the mesentery. This, according to Singer,[6] should arouse suspicion of Hodgkin's disease. The infiltration may either ulcerate or the cells in the submucosa may grow rapidly and result in the hyperplastic, firm type which is difficult to differentiate grossly from carcinoma. The microscopic appearance is that of a hyperplasia of the lymphoid reticulum in the submucosa; it is in this tissue that the plasma cells, eosinophiles and Dorothy Reed cells are found.

Clinically, the symptoms resemble those of carcinoma, that is, epigastric pain, eructation of gas, hematemesis, melena, loss in weight and strength are noted in both conditions. Rarely, as in our case, is a mass felt. The duration of symptoms is, for the most part, short; it is a matter of only a few weeks as in the case reported by Sherman,[7] three months in the case herein reported. In a case reported by Singer,[6] relief of pain was obtained for several

months by a Sippy diet. The laboratory and x-ray findings are also similar to those of carcinoma. A secondary anemia is usually found. The white blood count is usually low; rarely is an eosinophilia noted Koenig[8] and Culver, in 1941, reviewed the literature on intestinal Hodgkin's disease and reported three new cases with particular reference to x-ray findings and concluded that there were no roentgen differences between Hodgkin's disease and other infiltrative, ulcerative lesions of the stomach. Jungmann[9] and others, however, have pointed out that in spite of the degree and extent of stomach involvement, peristalsis can still be seen in the stomach on x-ray examination and believed that this might be used to distinguish Hodgkin's disease from carcinoma.

From a review of the reported cases one concludes that the best plan of treatment is resection of the lesion either with or without postoperative x-ray therapy. The first case of successful resection was reported by Steindl[10] in 1924. When surgery can not be done, roentgen therapy may be used to lessen the severity of the symptoms.

CONCLUSIONS

1. A case of Hodgkin's disease limited to the stomach is reported. The patient is active thirteen years after resection of the lesion.

2. All masses or indurated ulcers of the stomach are not cancerous. Hodgkin's disease should be considered in differential diagnosis.

3. Resection of the lesion gives a good chance of cure.

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SPONTANEOUS HEALING OF AN EXTERNAL BILIARY FISTULA

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FISTULAS of gallbladders or of the biliary ducts are divided into internal and external fistulas. In internal biliary fistulas the fistular tract unites the gallbladder or a biliary duct to an internal abdominal organ. There are fistulas connecting the biliary tract to any portion of the gastrointestinal canal, to the kidney, to the urinary bladder or to the uterus. There are also reported cases of fistulas between the choledochus and the portal vein.

In the external fistulas the biliary ducts are connected with the external surface of the abdominal walls. Internal fistulas are due to adhesions and ulcerations of the two viscera and are found in about 5 per cent of all patients who are to be operated upon for diseases of the biliary tract.

The frequency of internal fistulas has remained unchanged but the external fistulas, generally the sequel of abdominal operations, are not found now so frequently as before in consequence of the progress and improvement in the operative technic obtained in recent years. While Louis Courvoisier was able, in 1890, to collect and classify 169 observations of external biliary fistulas, probably today there are surgeons who never have seen a single case of external biliary fistula consecutive to operation on the biliary tract.

External biliary fistulas very rarely heal spontaneously but generally require an operation. The operation consists in changing the external fistula into an internal one by connecting the fistular tract to the stomach or another part of the intestinal tract. Since such an operation is always difficult and often unsuccessful, I think it may be of interest to report a case of spontaneous healing of an external biliary fistula and see if we can explain what the conditions are that favor such a rare and fortunate event.

CASE REPORT

On June 13, 1938, a female patient, single, aged thirty-eight years, was admitted to the author's service at Columbus Hospital with the diagnosis of acute calculous cholecystitis. On June 30th, she was operated upon by the writer; cholecystostomy was performed numerous calculi were removed and the gallbladder was drained.

On August 3, 1938, the patient was discharged from the hospital in an improved condition. For a few months after the author had occasion to examine the patient frequently and he found her wound apparently perfectly healed. For many years he had no occasion to see or hear of the patient, but almost eight years later, on April 5, 1946, the patient was readmitted to Columbus Hospital affected with abscess of the liver. She was operated upon on April 17th with incision and drainage of a large abscess of the left lobe of the liver. On June 26, 1946, the patient was discharged from the hospital affected with an external biliary fistula.

After leaving the hospital and for more than three months there was a continuous leakage of bile from the fistular opening, at times in such a quantity as to seem a real outpouring. However, her general condition was good and the stools always contained bile.

She was treated at home by the writer up until October, 1946, when having seen no healing he decided that it would be necessary to operate, to dissect the fistular tract and connect it to the gastrointestinal canal. She was readmitted to the hospital on October 9th. It was intended that we should keep her under observation for a few days to prepare her for operation. The day after admission we were surprised to find that the fistular opening was closed. She was discharged from the hospital on October 16th apparently healed. The fistular opening remained closed and after a few days of rest, the patient's condition was so that good

she was able to resume her work in the factory where she had been employed and to take care of her household duties. She is perfectly well now.

In the literature of external biliary fistulas there are reported cases of healing of such fistulas without operation after treatment with vitamin K, with curetting of fistular sinuses and with the use of ultra-violet rays, but in this case the only treatment applied to the fistula was the ordinary dressings to keep the surroundings of the fistular opening clean.

With repeated x-ray examinations, we found that no calculus was present in the fistular tract or in its vicinity and by frequent inspection we always made sure that bile was passing into the intestinal canal, that the stools always showed the presence of bile and we trusted that with its natural coarctation and narrowing the fistular tract would in the end close.

In conclusion it should be stressed that although the greater number of external biliary fistulas require a surgical operation, a small number of them can heal spontaneously without any operation. The most important sign that a spontaneous healing could possibly be expected is if some bile enters in the intestinal tract and is revealed by examination of the stools. When no calculus is seen and the presence of bile is demonstrated in the stools, it is worth while for the patient to wait and for the surgeon to delay operation for a reasonable time. If spontaneous healing occurs, the patient will be amply rewarded for the inconveniences resulting from enduring the fistula a few weeks longer.

New Instruments

METACARPAL FRACTURES

MODIFICATION OF A NEW INSTRUMENT FOR THE MAINTENANCE OF POSITION AFTER REDUCTION

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THE writer has recently described a new instrument for the treatment of metacarpal fractures.[1] This instrument was devised for the simple transverse fractures involving the lateral four metacarpal bones and uses the principle of pressure pads to maintain position of the fragments after closed reduction.

Essentially this method of treatment, in part, was based on the principle originally advocated by Jahss[2] in so far that it uses the flexed proximal phalanx to push upward on the small distal fragment. Unlike the principle of Jahss the proximal fragment is held down and not allowed to go through its full physiologic excursion until it is anchored by the carpometacarpal joint capsule. Thus, the amount of upward force on the flexed proximal interphalangeal joint is materially reduced. Unlike the method of Jahss the pressure pads are adjustable, allowing the operator to regulate frequently the pressure and daily extend the proximal interphalangeal joint. This eliminates its greatest difficulty, namely, a pressure sore or flexion deformity at this joint.

The neck of the metacarpal which is the smallest and weakest portion of the bone is the most common site of the fracture. The fracture is more frequently transverse in character. The resulting deformity is that of a depression of the head with drosal angulation of the fragments. The more dorsal the striking force against the head the greater is the depression of the knuckle. The more direct the force on the head in the longitudinal axis of the metacarpal the greater the degree of impaction. A tangential force results in depression of the head with impaction and angulation of the fragments.

Normally the interosseous and lumbrical muscles insert into a loose aponeurotic sleeve over the dorsum of the proximal phalanx just distal to the metacarpophalangeal joint. (Fig. 1.) Contracture of these two muscles results in flexion at the metacarpophalangeal joint.

Following a fracture of the metacarpal with depression of the metacarpal head, the directional pull of these muscles is altered and the proximal phalanx is pulled into hyperextension. (Fig. 2.) The greater the flexion deformity of the metacarpal, the more will be the hyperextension pull of the lumbrical and interosseous muscles, the shorter will be the relative length of the flexor profundus and sublimis tendons and the greater will be the flexion deformity of the interphalangeal joints.

This deformity in the laborer interferes with his grasp of an implement. The inability to flex the affected finger completely results in weakness of the flexion power of the adjacent fingers. The prominent head of the metacarpal in the palm of the hand may cause pain when a object such as a tool is grasped. Depression of the knuckle, prominence on the dorsum of the hand, hyperextension at the metacarpophalangeal joint and flexion deformity at the interphalangeal joints are cosmetically objectionable to women.

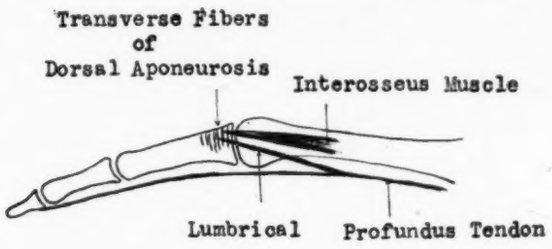
Unfortunately in this fracture which is so easily reduced it is difficult ordinarily to

maintain the corrected position. Since this type of fracture is usually first seen by the general practitioner, it is invariably treated by him with one of the many more simple methods available. These usually include the ball or roll of bandage splinted in the palm of the hand or various types of plaster of paris, wooden or leather splints. Surgeons have used more complicated methods: Bosworth[3] maintains position by fixing the fragments with rigid wires into the adjacent metacarpal, Meltzer[4] employs the skeletal wire traction through the proximal phalanx, Carr[5] maintains the position of the fragments by inserting specially constructed ice tongs into the proximal phalanx and Bunnell[6] uses longitudinal and transverse Kirschner wires.

The ball or roll of gauze in the palm of the hand does not counteract but rather emphasizes the angulating pull of the lumbrical and interosseous muscles thereby increasing the deformity. Skeletal traction of any form or skeletal fixation introduces the danger of infection. When plaster is used, it is too difficult to determine the exact amount of pressure necessary to maintain the position of the fragments. Too much pressure will result in pressure sores during the swelling stage which ensues within a few hours following reduction. Too little pressure will result in reangulation when the swelling subsides. Constant immobilization of the flexed proximal interphalangeal joint, as advocated by Jahss, for a period sufficient to see callous on the x-ray film often results in a flexion deformity.

With the fracture once reduced, the operator will find that very little upward pressure on the head of the metacarpal and downward pressure over the distal end of the proximal fragment is necessary to maintain the corrected position. However, an upward force cannot be made at this time on the plantar surface of the metacarpal head since pressure of any consequence on the intervening flexor tendon sheath would irritate its membrane and result in thickening and adhesions. In order to eliminate pressure over the flexor tendon sheath the metacarpophalangeal and first interphalangeal joints are flexed to form right angles. This places the base of the proximal phalanx beneath the head of the metacarpal. Slight pressure exerted upward against the head of the flexed phalanx results in elevation of the metacarpal head and easily maintains the corrected position of the fragments. The amount of pressure necessary is merely the equivalent of overcoming the angulation deformity caused by the pull of the interosseous and lumbrical muscles.

The instrument originally described has been in use by the author and also by his associates since 1941 with equally good results. These fractures were originally treated with a metal splint using the flexed finger throughout the entire period of im-



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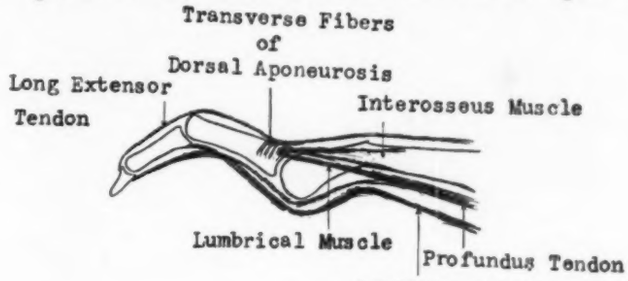
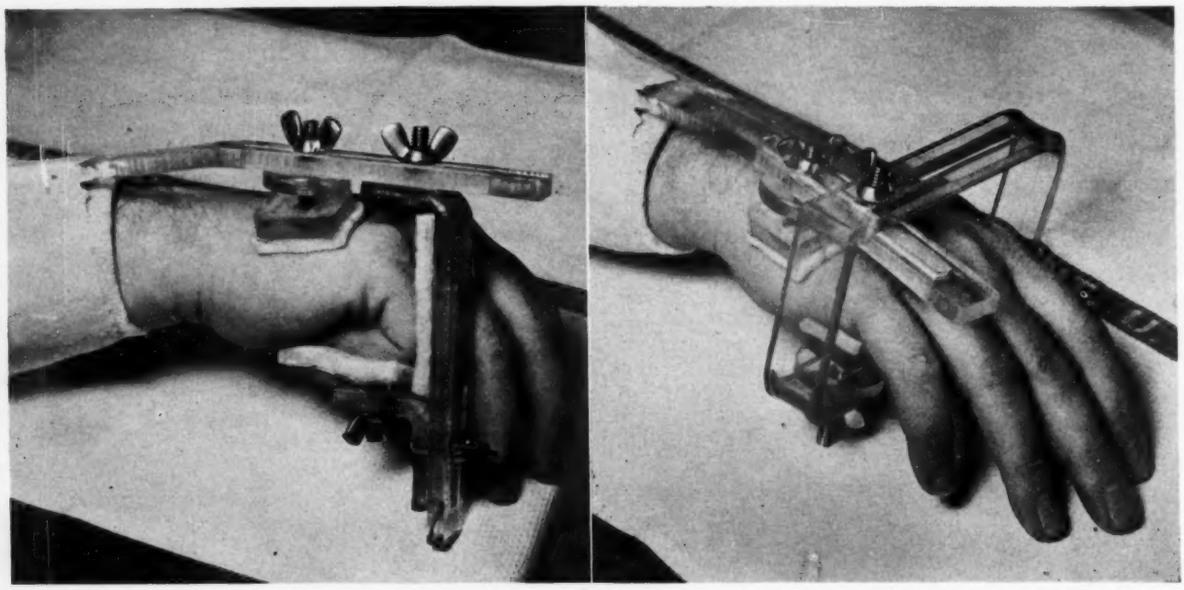


2

Fig. 1. A schematic drawing demonstrating the normal relationship of the interosseous, lumbrical and profundus muscles.

Fig. 2. A schematic drawing demonstrating the relationship of the intrinsic muscles after a flexion deformity of the metacarpal has occurred. The force of the lumbrical and interosseous muscles have been altered from a ventral to a dorsal pull. Because of the hyperextension deformity at the metacarpophalangeal joint, the sublimis and profundus tendons have become relatively shortened, thus causing a flexion deformity at the distal interphalangeal joints.



A B

FIG. 3. A and B, the original and the modified splints incorporated. Both splints can now be used throughout the entire course of treatment. A is used during the first three or four days of treatment. The distal attachment is then removed by a simple bolt and the square attachment applied. This allows the involved finger to be extended during the remaining period of immobilization.

mobilization. (Fig. 3A.) At that time it was necessary to see the patient every second or third day in order to extend the proximal interphalangeal joint and thus avoid a flexion contracture. An attempt was then made to eliminate the necessity of seeing the patient so frequently.

After the first three to five days the swelling of the soft tissues and the spasm of the traumatized interosseous and lumbrical muscles had subsided. Therefore, it was believed that a slight amount of upward pressure on the ventral surface of the metacarpal head at this time would not irritate the flexor tendon sheath. A square attachment (Fig. 3B) was then devised which applied the upward pressure directly beneath the metacarpal head. This proved to be satisfactory but one difficulty was encountered. The splint which was constructed of metal obstructed adequate visualization of the fragments on the roentgenogram. Hence, on a subsequent case the splint was constructed of a plastic material and proved more satisfactory since it was radiolucent.

An attempt was then made to combine both instruments into one in order to simplify replacement of the vertical arm with the square attachment. This was accomplished by using a simple bolt to disconnect the vertical arm and replace it with the square attachment. Roentgenograms of a typical case are demonstrated. (Fig. 4A to D.)

TREATMENT OF A SIMPLE IMPACTED FRACTURE

The hematoma at the fracture site is injected with about 5 cc. of a 1 per cent solution of procaine hydrochloride. (Fig. 5.) Any skin glue such as compound tincture of benzoin, gum mastic or Ace adherent is applied to the distal half of the forearm and the glued area is now covered with a 2 or 3 inch circular stockinette. After ten or fifteen minutes the fracture site will have become anesthetized and the fragments are forcibly broken up to disengage the impaction. *This is absolutely imperative.*

The fragments are easily aligned by applying upward pressure on the palmar surface of the metacarpal head and downward pressure over the distal portion of the proximal fragment. The patient now maintains the corrected position by placing the

FIG. 4. A, simple fracture through the neck of the fifth metacarpal; B, fracture reduced and the original splint applied.

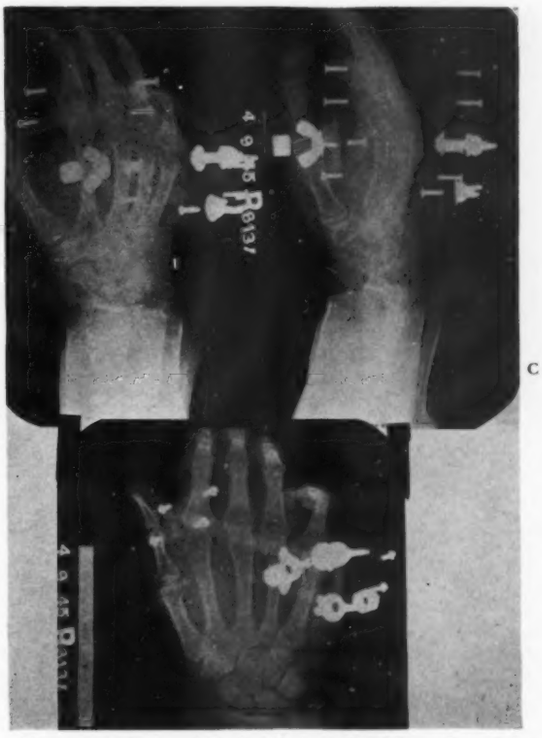


FIG. 4. C, the modified splint applied four days later shows the position of the fragments maintained.

FIG. 4. D, twenty-six days postreduction; fragments in good position; motion of fifth finger normal.

index finger of his other hand beneath the metacarpal head and his thumb over the dorsum of the hand just proximal to the fracture site. The operator then applies a few turns of plaster of paris bandage about the forearm over the stockinette. The long arm of the splint (Fig. 3) is then applied in the line of the involved metacarpal and anchored to the forearm with another few turns of plaster. In a few moments the

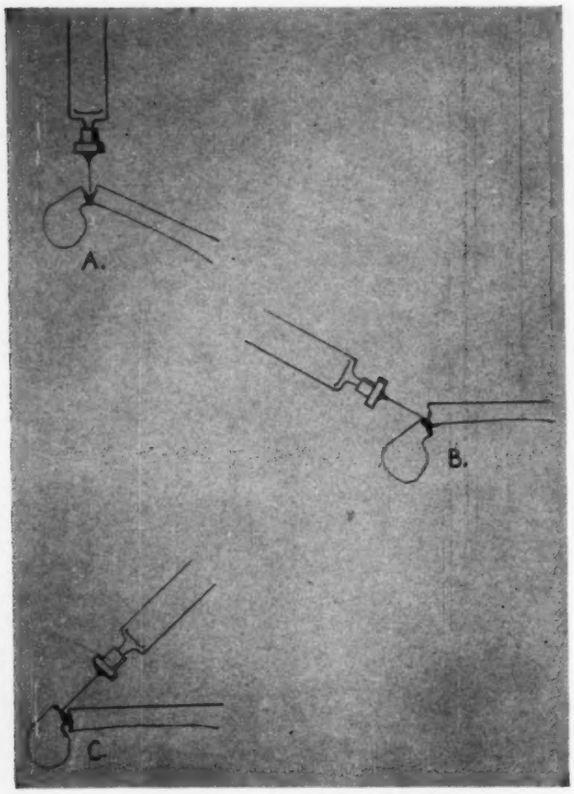


Fig. 5. A schematic drawing demonstrating the various directions of the hypodermic needle to facilitate striking the hematoma at the fracture site. At this point about 5 to 10 cc. of a 1 per cent solution of novocain is injected in order to produce analgesia.

plaster will have set. The upper set screw is then adjusted so that the pressure plate rests directly over the metacarpal and is proximal to the fracture line. The involved finger is then flexed to right angles at the metacarpophalangeal and proximal interphalangeal joints. The patient now exerts upward pressure on the distal fragment by pressing upward on the flexed proximal interphalangeal joint and downward on the dorsal pressure pad. The vertical arm is now attached to the long horizontal arm by means of a bolt and nut. (Figs. 3A and 6.) The lower support bracket is raised so that it approximates the dorsal surface of the flexed second and third phalanges. The knuckle is thus maintained in its normal prominent position.

If the fifth metacarpal is involved, it will be noticed that the two terminal phalanges will deviate radially. Hence, a third set screw is placed at the lower support in order to adjust it to the proper angle. The upper plate is supported by a universal joint in order to allow for slight variations in the size and shape of the hand and still maintain an even distribution of force throughout the entire contact surface which is covered with a felt pad. To prevent the possibility of rotation of the fragments it is advisable that the next normal finger be flexed with the involved finger and then released either immediately or after one or two days. The lower pad can be placed transversely to accommodate both flexed fingers. It can also be angulated to compensate for the different lengths of the proximal phalanges.

First Postoperative Day. Twenty-four hours after reduction the operator's thumb can be placed beneath the involved metacarpal head and his index finger over the upper pressure pad. With the position maintained in the aforementioned manner, the lower pad is released and the flexed finger carried through a full range of motion at the proximal interphalangeal joint. The finger is lightly massaged with any oil, flexed again and the lower pressure pad reapplied. This procedure is carried out daily for three to five days to eliminate the possibility of a pressure sore and flexion deformity at the proximal interphalangeal joint.

Square Attachment. After three to five days the swelling of the hand and the spasm of the lumbrical and interosseous muscles, which is the deformity element, will have subsided. The fragments are again temporarily held by the patient or the operator. The vertical attachment is removed and the square attachment is applied. (Fig. 3A and B.) Pressure pads are then adjusted (Figs. 3 and 7) and in this manner the patient had practically a full range of finger motion. It is now necessary to see the patient only once or twice weekly until callous is visible at the fracture site which usually takes three to five weeks. The splint is then removed and no further treatment is necessary.

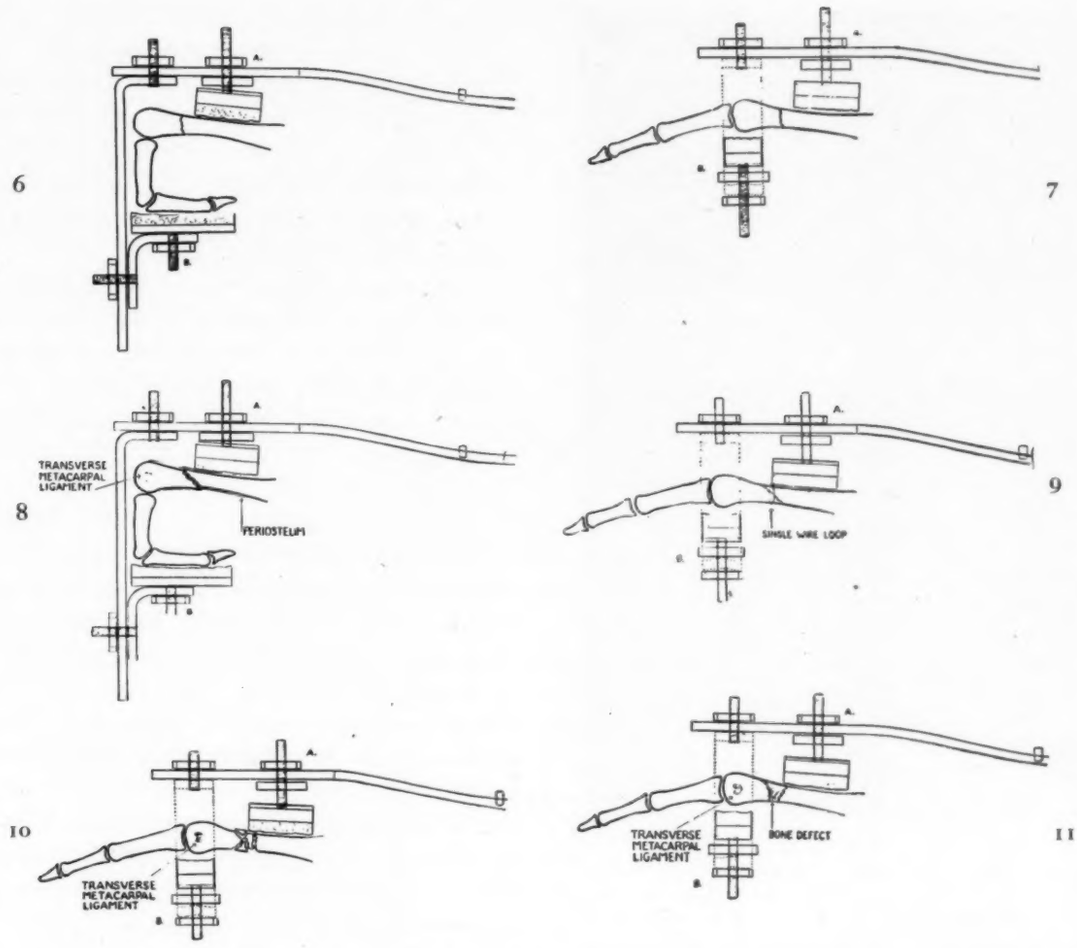


Fig. 6. The vertical attachment applied to a simple fracture through the neck of a metacarpal. Pressure at "A" and "B" applied through the intervening extensor tendon during the variable phases of swelling and muscle spasm following reduction.

Fig. 7. The square attachment applied to a simple fracture through the neck of a metacarpal. This attachment is applied three to five days after closed reduction. Pressure now at pad "B" is insufficient to cause injury to the intervening flexor tendon sheath.

Fig. 8. The vertical attachment applied to a simple oblique or spiral fracture through the shaft of a metacarpal. When reduced, the "saw-tooth" edges of the fragments, the "sleeve-like" effect of the periosteum and the "anchorage" of the transverse metacarpal ligament to the adjacent metacarpal may be sufficient to prevent over-riding while the splint maintains position.

Fig. 9. The square attachment applied to a simple oblique or spiral fracture through the shaft of a metacarpal. If the fragments slip after closed reduction, a single wire loop can be inserted into a small drill hole under local anesthesia. The wire loop tends to overcome some of the angulating pull of the lumbricals and interosseous muscles.

Fig. 10. The square attachment applied to a comminuted fracture through the shaft of a metacarpal. The anchorage of the transverse metacarpal ligament to the adjacent metacarpal prevents marked shortening. Good alignment in spite of little shortening leaves a satisfactory functional result. Pressure at "B" is lessened since the slight bone shortening diminished the angulating force of the lumbrical and interosseous muscles.

Fig. 11. The square attachment applied to a compounded war injury with a bony defect. The splint holds the fragments in good alignment until the soft tissue wounds heal. The pressure pad at "A" may easily be released when change of dressing is necessary. Apparatus may be reapplied after bone graft is inserted.

FIG. 12. A, malunited fracture near base of third metacarpal with palmar angulation of distal fragment; bullet wound first seen three months after injury. B, the deformed fifth finger was disarticulated at the proximal interphalangeal joint; the middle phalanx was used as a bone graft; open operation performed on third metacarpal which was united by fibrous union; graft held *in situ* with silver wires; fragments maintained in good position three weeks postoperatively.

HANDLING OF OTHER FRACTURES

Transverse or Oblique Fractures. Occasionally the periosteum, the anchorage of the transverse metacarpal ligament to the adjusted metacarpal and the "saw-tooth" shape of the fractured fragments will hold them from over-riding. In that case the fragments can be immobilized in the usual manner. (Fig. 8.) On the other hand, if the fragments slip, it is a simple procedure to approach the fragments through a small dorsal incision under local anesthesia and insert a single wire loop through a very small drill hole in each fragment. The wire loop counteracts some of the angulating pull of the spastic lumbrical and interosseous muscles hence obviates the necessity of using the vertical attachment. Therefore, the square attachment may be applied immediately after reduction. The upper pad is placed directly over a small dressing which can be held in place with any skin glue. (Fig. 9.)

Compound Fractures. The wounds can be cleaned and dressed in the usual manner. Slight shortening of the metacarpal diminished the angulating pull of the spastic lumbrical and interosseous muscles. The square attachment may be directly applied. (Fig. 10.)

Fractures Caused by War Injuries. When bony defects are encountered as a result of bullet or shell fragment injuries, the wounds can be débrided and dressed in the usual manner. The square attachment is applied over the dressing. If the wound if left open, the fragments can be held by the operator while the pressure pads are removed and the wound can be redressed. The pressure pads are then reapplied. In this manner the fragments are held in good alignment until the soft tissues have healed. (Fig. 11.) By this time the intervening fibrous tissue will hold the fragments and prevent angulation. At some subsequent date an open operation can be performed, a small bone graft inserted, held *in situ* with a single wire loop at each end and the square attachment reapplied. During this entire procedure the patient has full motion of all his fingers.

Malunited Fractures. Open operation followed by osteotomy of the fragments is simply performed. The square attachment is then directly applied. If there is fear that the fragments might slip, then a single wire loop can be inserted into both fragments.

Ununited Fractures. The intervening fibrous tissue is excised. The fragments are freshened and a small bone graft may be applied. The graft can be held *in situ* with one or two small wire loops. (Fig. 12A and B.)

CONCLUSION

A simple mechanical device has been described for use in the treatment of all types of fractures involving the four lateral metacarpal bones.

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According to T. Horwitz, in congenital or developmental coxa vara, drilling of the femoral neck or implantation of a bone graft through it as a supplemental procedure to corrective osteotomy is unjustified. He tried these methods in a small series of cases but found them of no particular value. Any child with a painless limp may have this deformity. Pain later appears in adolescence or adult life as a result of increasing deformity, especially of the femoral neck, and shortening of the involved extremity. X-rays are diagnostic, of course. Simple subtrochanteric osteotomy (or intertrochanteric osteotomy) is followed by satisfactory results in most cases. Recurrence may occur especially in those with chronic cases who are operated upon too late, and undoubtedly secondary degenerative changes in the femoral head and neck will be found by x-rays in many of these instances. (*Richard A. Leonardo*, M.D.)

A THREE-WAY INFUSION VALVE FOR BIOLOGIC FLUIDS

WILLIAM BIERMAN, M.D.
New York, New York

THE valves employed in medical practice today appear to be direct adaptations of those used commercially for gases and liquids. The problems encountered when it is desired to transfer biologic material from one channel to another would indicate the necessity for a device which would be better suited for its purpose than the present commercially inspired valves.

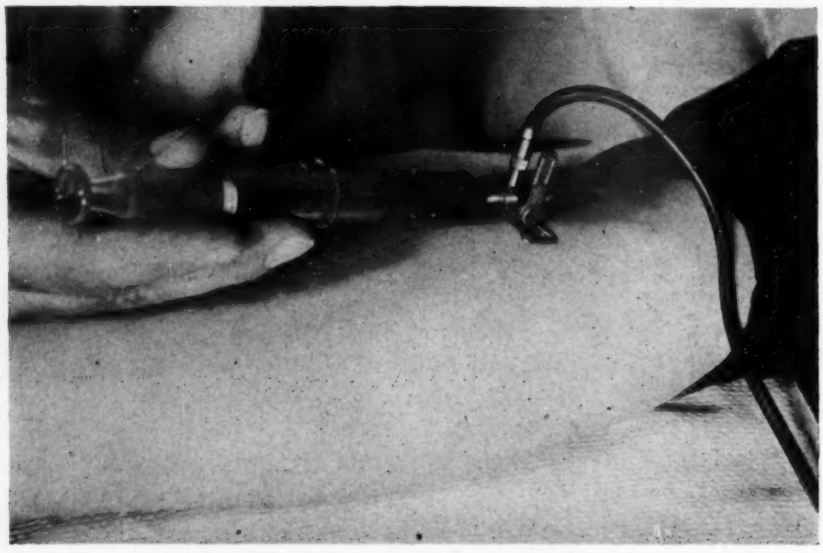

FIG. 1. Illustration of three-way infusion valve.

The author has accordingly devised a three-way metal valve designed to facilitate work with blood and with other biologic fluids. This valve has the great advantage that it can be readily taken apart for cleaning and can be reassembled with equal ease and speed. This, in addition to large channels, makes it a simple matter to remove material such as blood clots, fibrinous or other masses which might clog the instrument.

The valve, shown in the illustration, consists essentially of two sections, one a tapered member fitting easily into the opening of the other. These are held together snugly by an outside spring. The spring tension although firm is adjusted to allow the valve to be turned easily. The connections for syringe and adapters are of standard size. A short metal arm attached to the rotating portion of the valve points in the direction of the channel through which the fluid is flowing. Guiding pins placed on the side of the valve to limit the excursion of this metal arm cause exact alignment of the desired channel. This permits the operator to turn the valve by the sense of touch and without necessarily looking at it.

The tapered portion of the valve on to which the needle fits is placed eccentrically so as to allow the needle to be held close to the skin surface. The crosspiece attached to the under surface of the valve permits it to remain in place when employed for intravenous work in an extremity. If desired the valve with its connections can be stabilized further by stretching strips of adhesive from the crosspiece to the adjacent skin.

Acknowledgments: The author wishes to express his thanks to Dr. A. W. Shenker of New York and to Mr. Emil Davidson of the Clay Adams Co. for their assistance in the construction of models of this valve, and to Dr. Ivan Brown of Duke University Hospital for helpful suggestions and for the testing of this apparatus.

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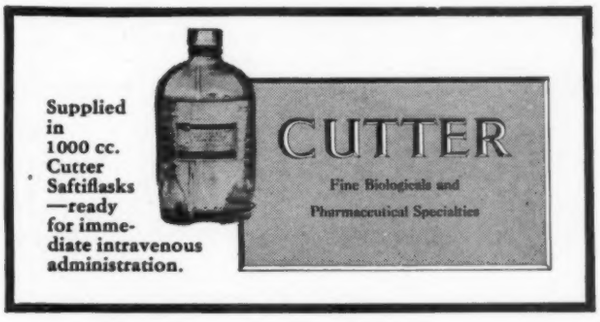
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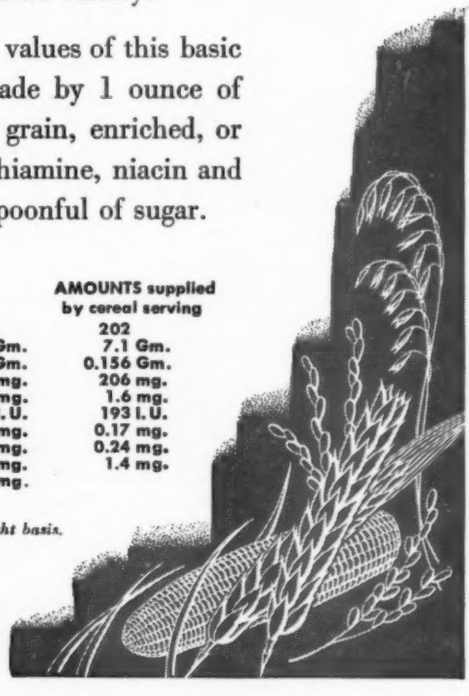

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Zymacaps

Outside the Ivory Tower

(*continued from page 24*)

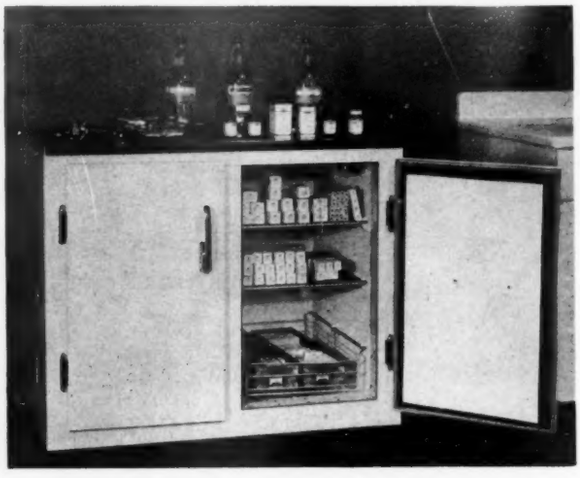
MANY miscarriages of early pregnancy take place because women do not possess a sufficiently high concentration of thyroid hormone in the blood, a Yale professor suggested today. The clue to many of these "spontaneous abortions" which thwart normal pregnancies can be found in the degree of concentration of precipitable iodine in the blood serum of a woman. The iodine is found in exceedingly small amounts in the blood but exerts a powerful influence. Non-pregnant women normally have a concentration of from 4 to 8 millionths of 1 per cent of this precipitable iodine. When they become pregnant, the concentration rises higher, ranging between 8 and 10 millionths of 1 per cent. In many cases in which pregnant women have failed to register such a rise miscarriages have occurred early in pregnancy. These facts, which throw new light on the century-old problem of miscarriages, were announced by Dr. John P. Peters, John Slade Ely Professor of Medicine at Yale.

ARTERIOSCLEROSIS or hardening of the arteries, a major factor in the appalling number of deaths due to diseases of the heart and blood vessels, is more to be feared than the atom bomb, according to Dr. Theodore G. Klumpp, president of Winthrop-Stearns, Inc.

HIGH prices have hit the hospitals such a staggering blow that many may have to close their doors in 1948. That "storm signal of approaching disaster" was announced recently by leaders of Manhattan's United Hospital Fund, who are waging a desperate campaign to save New York City's voluntary hospitals from financial collapse. In the past year, hospital payrolls jumped 40 per cent, food more than 43 per cent, drugs 38.9 per cent. Meanwhile, the hospitals had an unprecedented horde of patients. Result: most voluntary hospitals in the city went deeply into the red (*e.g.*, the immense New York Hospital had an operating deficit of $1,540,115). Even after drawing on endowments and gifts, the city's eighty-nine hospitals wound up with $2,400,000 in unpaid bills.

A CHILD born today can expect to live to be a hundred if he is given proper care in infancy and youth, and if he avails himself of present medical knowledge through his adult years, Dr. Edward L. Bortz, President of the American Medical Association, announced recently.

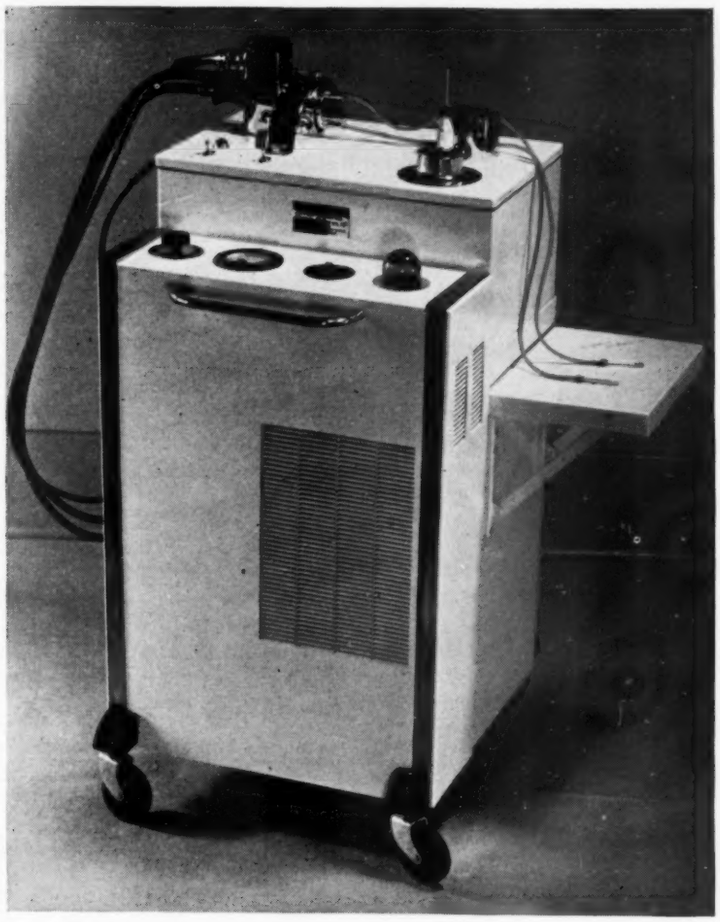
THE development by Dr. Philip Levine, one of the world's outstanding serologists, of a highly accurate diagnostic anti-Rh (anti-D) serum derived from human blood has been announced by the Ortho Pharmaceutical Corporation of Raritan, N.J., the first of the pharmaceutical houses to offer this type of serum for determining whether an individual has Rh negative or positive blood. It is now available for use by hospital and clinical laboratories to prevent intragroup transfusion accidents and for the selection of Rh negative blood for the affected infants of Rh negative mothers.



A REFRIGERATION unit suitable for use in the doctor's office is now being manufactured by Artcraft, Lima, Ohio. This unit offers space above for the compounding of prescriptions and below for controlling refrigeration of such drugs as penicillin, biological serums, etc. The lowboy is of table-top height with a stainless steel working surface 42 inches long and 23½ inches wide.

(*continued on page 40*)

KNOTT HEMO-IRRADIATOR



THE KNOTT
HEMO-IRRADIATOR

is endorsed by the American Blood Irradiation Society as the only apparatus enabling the uniform irradiation of such an opaque substance as blood and controls the exposure and other factors within the narrow range having the desired therapeutic values.

All published reports on this procedure are based on the use of the Knott Hemo-Irradiator in accordance with the established Knott Technique.

This procedure is reported in this publication under the title:

"DEVELOPMENT OF ULTRAVIOLET BLOOD IRRADIATION" Page 165

The following reprints and others upon request:

ULTRAVIOLET IRRADIATION OF BLOOD IN THE TREATMENT OF ESCHERICHIA COLI SEPTICEMIA; Archives of Physical Therapy, March 1943.
PRESENT STATUS OF ULTRAVIOLET BLOOD IRRADIATION (KNOTT TECHNIC); Archives of Physical Therapy, June 1944.
PREOPERATIVE HEMO-IRRADIATIONS; American Journal of Surgery, August 1943.
ULTRAVIOLET BLOOD IRRADIATION TREATMENT OF PELVIC CELLULITIS (KNOTT METHOD); The American Journal of Surgery, October 1947.
PYOGENIC INFECTIONS; American Journal of Surgery, September 1942.
ULTRAVIOLET BLOOD IRRADIATION IN BILIARY DISEASE (KNOTT METHOD); American Journal of Surgery, August 1946.
ULTRAVIOLET BLOOD IRRADIATION THERAPY IN ACUTE VIRUS AND VIRUS-LIKE INFECTIONS; The Review of Gastroenterology, April 1948.
ULTRAVIOLET IRRADIATION OF AUTOTRANSFUSED BLOOD IN THE TREATMENT OF POSTABORTIONAL SEPSIS; American Journal of Surgery, March 1942.
TREATMENT OF BLOOD STREAM INFECTIONS WITH HEMO-IRRADIATIONS; American Journal of Surgery, December 1942.
ULTRAVIOLET BLOOD IRRADIATION THERAPY (FURTHER STUDIES IN ACUTE INFECTIONS); American Journal of Surgery; April 1947.

SCIENTIFIC EQUIPMENT MANUFACTURING CO.

302 Prefontaine Building — Seattle 4, Washington

Outside the Ivory Tower

(*continued from page* 38)

FOR the first time since the Passano Foundation was established in 1944, a dual selection has been made. The award goes to the famous Taussig-Blalock team who developed the successful operative procedure which is popularly known as the "blue baby operation." It also marks the first time that a woman has been named as an award winner. The Williams & Wilkins Company, Medical Publishers of Baltimore, established the Passano Foundation to aid in any way possible the advancement of medical research, especially research which bears promise of clinical application. Previous winners have been Dr. E. J. Cohn, of Harvard, for his work on fractionation of blood; Dr. Ernest Goodpasture, of Vanderbilt, for virus culture by the chick embryo method; Dr. Selman Waksman, of Rutgers, for discovery of streptomycin.

USE of radioactive iodine from the atomic reaction piles at Oak Ridge, Tenn., in the effective treatment of toxic goiters, was described at a meeting of the experimental medicine section of the Academy of Medicine of Cleveland by a group of medical scientists from Western Reserve University and University Hospitals. They reported that during the past year they treated eighteen goiter victims and two-thirds were cured or definitely improved. It was indicated the others would improve on further treatment. There were no untoward reactions, except in one instance in which the patient contracted myxedema which could be counteracted by thyroid extract.

AUTHORITY has been granted to the Army Medical Department to place 300 additional Medical Service Reserve officers immediately on extended active duty, it was announced by Major General Raymond W. Bliss, The Army Surgeon General. Reserve officers who volunteer will be assigned to duty in all four sections of the Corps according to their qualifications. The greatest need is in the Medical Allied Science Section which includes personnel trained in psychology, physiology, biochemistry, nutrition, serology and other related medical sciences needed in the Army. The other three sections of the Medical Service Corps are the Pharmacy, Administrative and Supply Section, the Optometry Section and the Sanitary Engineering Section.

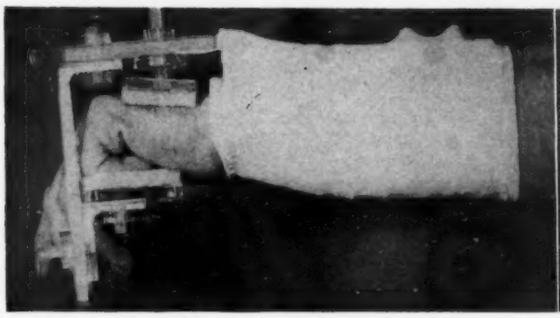
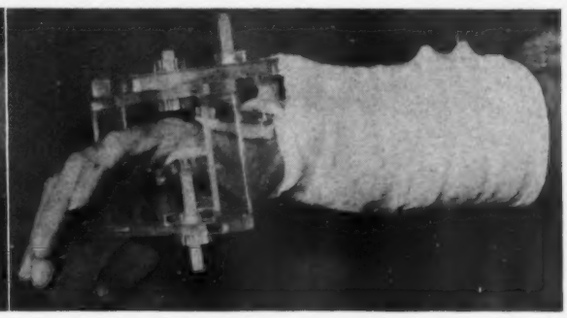
THE five leading causes of death during infancy, and the infant mortality rate in 1946 were: premature birth, 12.1; congenital malformations, 4.5; pneumonia and influenza (combined), 3.8; injury at birth, 3.6; diarrhea, enteritis and ulceration of the intestines (combined), 1.7. Almost three-fourths of all infant deaths occur within one month of birth. The first days are particularly hazardous. In 1946 33.9 per cent of all deaths occurred under one day of age, 25.6 per cent during the remainder of the first week and 11.7 per cent during the second, third and fourth weeks.

THE enrollment of 417,191 persons in Associated Hospital Service—New York's Blue Cross Plan—during 1947 brought the overall total to 3,206,178, or one out of every three persons in the Greater New York area.

DR. ROBIN C. BUERKI, dean of the Graduate School of Medicine and director of hospitals at the University of Pennsylvania, has been elected vice-president of the University in charge of medical affairs, it was announced by Dr. George Wm. McClelland, president of the University.

THE continuation of promising research into the causes and treatment of alcoholism is assured by a grant of $30,000 from The Research Council on Problems of Alcohol to the Cornell University Medical College.

DR. JOHN H. GARLOCK in the *New York State J. of Medicine* states that, "The indiscriminate use of penicillin in the treatment of certain surgical infections may mask the true progress of the pathologic state for which it is being given. After penicillin treatment for more than 48 hours, 2 patients with acute appendicitis gave every evidence of a perforated lesion with diffusing peritonitis, yet did not seem as acutely ill as is usual in this latter stage of the disease."

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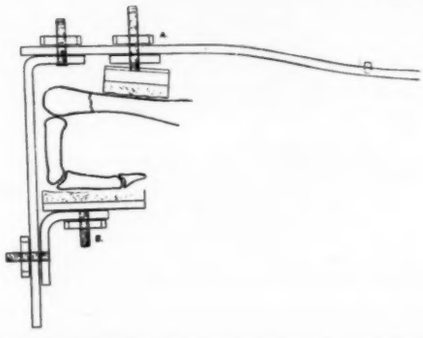
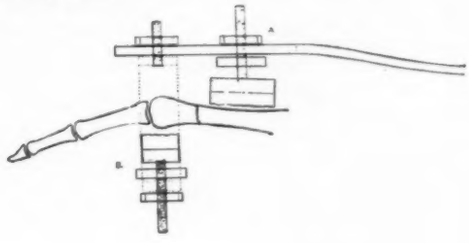

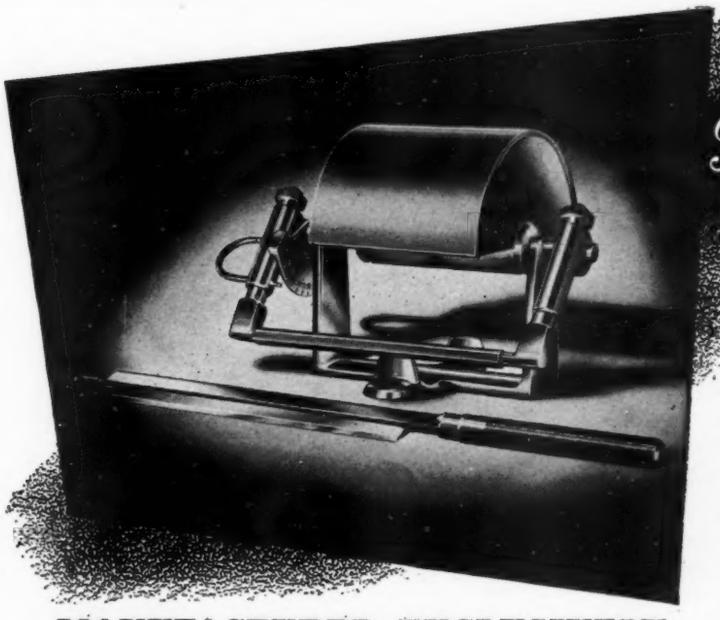

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● By means of this unique instrument a sheet of skin of uniform thickness may be cut at a predetermined level from any area of the body as large as 4 x 8 inches (the size of the drum). Those who have used the Dermatome state that it simplifies the technique and considerably enlarges the whole field of skin grafting. The Dermatome blade may be inserted in the handle for honing the blade and also for cutting razor grafts. Rubber cement for the drum and donor area is available, extra knives are kept in stock, and a knife sharpening service is maintained. Carrying cases are available upon request.

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Right away they want to know what a 'slicker' has to do with blood levels. The details go like this:

Hypercillin contains 300,000 units per cc. of 120 mgm. procaine penicillin G crystals, dispersed in fluid sesame oil with 2% aluminum monostearate, to delay absorption. This dispersing agent 'coats' the crystals, holding them in suspension for prolonged periods—and making smoother injections by cutting down needle plugging.

After injection—considerably less painful because of the procaine—the 'coated crystals' (water-proofed in their slickers) act as oily globules in a watery environment, where they are slowly absorbed. Absorption takes place around the periphery of the globule, and the procaine penicillin salt is slowly released into the blood —for therapeutic levels of at least 24 hours.

Hypercillin has two more factors of importance. The crystal size is large enough to maintain prolonged adequate levels—yet small enough to clear an 18-20 gauge needle without plugging. Sesame oil—the suspending medium—has these clinically established advantages: (1) less irritating to tissue; (2) less antigenic; (3) more suitable physically and chemically as a suspending medium.

Only Cutter Hypercillin offers a suspension of crystalline procaine penicillin G dispersed in sesame oil with 2% aluminum monostearate.

So you see why so many of my physicians are trying Hypercillin —and are agreeing "it's a slicker!"

Your CDM

(Cutter Detail Man)

*Trade Name for Cutter Procaine Penicillin G in Sesame Oil with 2% Aluminum Monostearate

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• Diagnosis is the art or scientific process by which a disease is recognized. Success or failure in establishing a correct diagnosis depends in large measure on the use of reliable, safe and efficient clinical and laboratory procedures.

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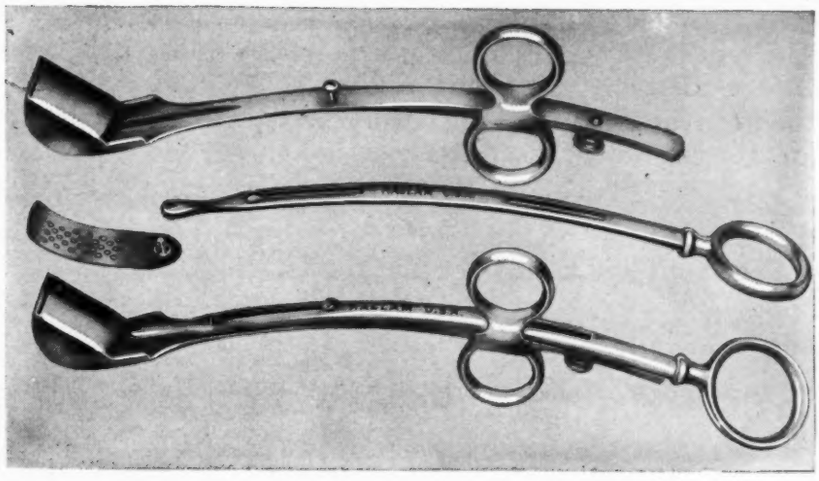

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LA FORCE ADENOTOME

The complete instrument is shown above in the bottom view, component parts are illustrated in upper part of cut. This instrument is available in three sizes, small, medium and large with one extra blade. Catalogue No. C 3950 R

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The Time Factor In Protein Nutrition

Although it has long been recognized that the dietary must contain appreciable amounts of biologically complete protein, only recently has the importance of simultaneous availability of the essential amino acids been established. In the light of recent investigations,* failure to provide the organism with the ten essential amino acids *at the same time* results in inability to synthesize protein, and makes positive nitrogen balance virtually impossible despite what may be regarded as adequate amounts of protein in the dietary.

These studies disclosed that tissue synthesis in protein-depleted rats proceeded rapidly when the basal ration was supplemented with the ten indispensable amino acids. When, however, these ten amino acids were divided into two groups, the first containing five and the second group the remaining five amino acids, alternate feedings with groups I and II at intervals as short as two hours produced startling results: the rats either continued on their downward nutritional trend or could barely maintain their weight. These experiments indicate that ingestion and absorption of all the essential amino acids must be synchronous for optimal utilization in tissue synthesis, and that protein waste will occur if ingestion is not properly timed.

In the light of these observations the presence of high quality protein foods in the daily dietary gains new significance, because biologically complete protein permits simultaneous utilization of all essential amino acids, making for most efficient nitrogen metabolism. The inclusion of complete protein in the dietary gives absolute assurance of simultaneous availability of all essential amino acids.

Meat is man's preferred source of biologically complete protein, capable—regardless of cut or kind—of improving the quality of less complete proteins supplied by many other foods.

*Cannon, P.R.; Steffee, C.H.; Frazier, L.E.; Rowley, D.A., and Stepto, R.C.: The Influence of Time of Ingestion of Essential Amino Acids upon Utilization in Tissue-Synthesis, Federation Proc. 6:390, 1947.

Cannon, P.R.: Amino Acid Utilization in the Surgical Patient, J.A.M.A. *135*:1043 (Dec. 20) 1947.

The Seal of Acceptance denotes that the nutritional statements made in this advertisement are acceptable to the Council on Foods and Nutrition of the American Medical Association.

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For the Successful Treatment of . . .

LEG ULCERS

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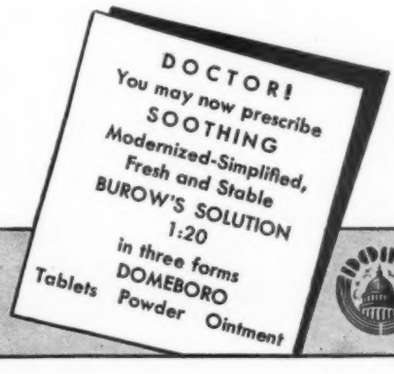
The technique developed by Dr. Cooper for the successful treatment of leg ulcers is based upon a 3 point program:

1. Reduce the associated Dermatitis by means of wet dressings of Domeboro Tabs (1:20 Burow's Solution).
2. Combat local infection and stimulate healing by a thick application of Daxalan in the center of the ulcer and surrounding area.
3. Overcome venous insufficiency, stasis and edema by wrapping Dome Paste Bandage around the entire leg to supply compression.

Daxalan is our trademark for a rigidly standardized whole crude coal tar paste (low in naphthalene content), uniform in color, free of coal tar specks and aged for six months.

Dome Paste Bandage is a flesh colored, 4" x 10 yd. gauze bandage impregnated with a modified "Unna's Formula" consisting of zinc oxide, glycerine, gelatine and calamine. This Unna's Boot comes to you in a soft condition and is ready for instant use.

Write for samples and literature



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Physician's package and complete description of the *New Technique* will be sent upon request.

Ethically promoted—Advertised only to the medical profession.

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Easily Fitted—The Lanteen Flat Spring Diaphragm, collapsible in one plane only, is easily placed without an inserter.

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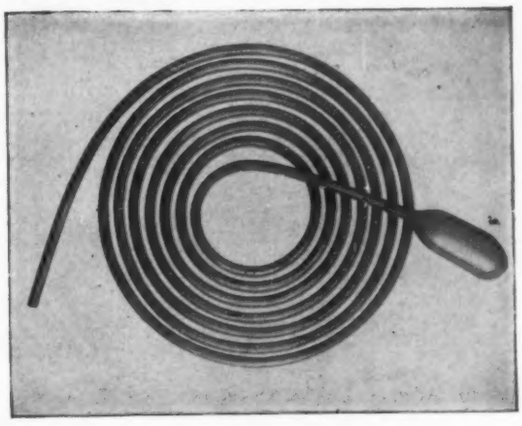

THE CANTOR TUBE is a bag-tipped, mercury weighted, single lumen tube. It is 18 Fr. and 10 feet long. Child Size Tube is 12 Fr., and 7 feet long. Its movement down the alimentary tract is actuated by a combination of free-flowing qualities of the mercury and the peristaltic action on the bolus formed by the mercury in the bag. Mercury is given the maximum motility by the loose bag attached distal to the tube. It is the only tube utilizing all the physical properties of mercury.

Adult Size Tubes are marked as follows to indicate their position: "S" for stomach at the 17″ mark, "P" for pylorus at the 24″ mark, "D" for duodenum at the 30″ mark, then in feet at the 4, 5, 6, 7, 8 and 9 feet marks. Child Size Tubes are marked as follows to indicate their position: "S" for stomach at the 14″ mark, "P" for pylorus at the 19″ mark, "D" for duodenum at the 24″ mark.

Secondary dilatation of the stomach can be decompressed by withdrawing the tube a short distance, cutting holes into the tube, and allowing the tube to be pulled down by peristalsis at which point the holes will open to the stomach which, on applying suction, will be decompressed.

Replacement bags are easily cemented to the tube.

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1. **Greater ease of intubation—first, ease of passage thru the nares and nasopharynx; and second, ease of passage thru the pylorus. Of 100 cases 96% were successfully intubated.**
2. **More efficient decompression—resulting from larger luminal diameter and less possibility of plugging.**
3. **Complete absence of any metal parts which might injure the mucosa.**

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D-111/B BAG for Child Size Cantor Intestinal Decompression Tube, with instructions for replacement of bag. Each $.60 Dozen $6.00

Ref: Described by Dr. Meyer O. Cantor, Detroit, American Journal of Surgery, July 1946, April and June 1947, March 1948

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The American Journal of Surgery

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two antihistaminics effective in allergies

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ANTISTINE

". . . a trial of both drugs increased the total percentage obtaining symptomatic relief to 90.2 per cent."[1]

Both Pyribenzamine and Antistine are effective antihistaminics and both are relatively free of side reactions.

A trial of these two drugs, determining which is most effective for the individual patient, will produce a far higher percentage of cases relieved than is possible with any single antihistaminic.

Even in vasomotor rhinitis (one of the more refractory conditions) this method has relieved 90.2 per cent of patients.[1] This far surpasses any previously reported response to antihistaminic therapy.

1. FRIEDLAENDER and FRIEDLAENDER: *Ann. of Allergy*, 6: 23, 1948.

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Ointment† and Cream,† 2%.

ANTISTINE† – Scored Tablets, 100 mg.
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*Council-accepted
†Not Council-accepted

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